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MANITOBA HAZARDOUS WASTE MANAGEMENT CORPORATION

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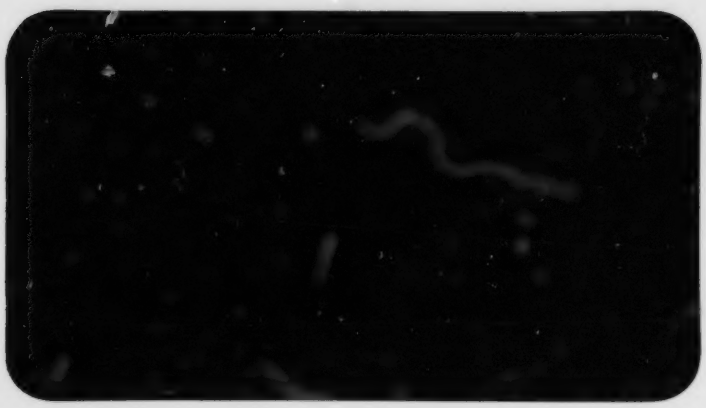
Corporate Profile The Manitoba Hazardous Waste Management Corporation is a commercial Crown corporation established to develop and operate a hazardous waste management system for Manitoba. Its mandate is to provide for the handling of regulated hazardous wastes from their source through to their treatment and disposal, consistent with high standards of public health, safety and environmental quality.



Environmental Policy Statement

The Manitoba Hazardous Waste Management Corporation is committed to the preservation and enhancement of environmental quality through the development of a hazardous waste management system for the province. This undertaking is a fundamental element needed to achieve the broader objectives of universal environmental stewardship and sustainable economic development within Manitoba.

To reach these goals, the Corporation uses the following policy objectives as the basis for the performance of its work.

- Minimize waste generation and maximize resource conservation through the promotion and facilitation of waste reduction, recycling, re-use and recovery at source.
- Ensure that all activities involved with the handling, storage, treatment and disposal of waste and residue products are consistent with the protection of public health and environmental quality.
- Earn public approval of the system and its components through open and responsive communication and through public participation in all aspects of their development and operation.
- Encourage economic development through the provision of a cost-effective waste management system and the enhancement of environmentally based enterprises.
- Foster and promote contemporary environmental values and policies through public education and research, as well as by example.
- Assume, in perpetuity, the environmental stewardship of wastes and residues placed in the Corporation's custody.

Information regarding the activities and programs of the
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HEALTH CONCERNS

AND

HAZARDOUS WASTE

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March, 1990

HEALTH CONCERNS AND HAZARDOUS WASTE

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EXECUTIVE SUMMARY

Objectives:

This report was produced under contract with the Manitoba Hazardous Waste Management Corporation (HWMC). The text of the scope of the work agreement is contained in Appendix 1. The objectives of this study are:

1. To critically review world epidemiological and public health literature to identify cases of suspected or substantiated claims of public health impacts associated with hazardous waste management facilities and potential products or emissions from such facilities; and

2. To place the above into perspective with respect to the development of a hazardous waste management system in Manitoba.

Methods:

Literature reviews were conducted through: (1) reviewing existing books (including Grisham, 1986; Andelman and Underhill, 1987; Travis and Cook, 1989) and summary reports (including the Environ Corporation report, 1986); (2) literature searches were conducted through Medline, Toxline, CISILO, and NIOSHTIC; (3) additional holdings by the HWMC and concerned citizens groups were gathered; and (4) letters were written and telephone contact established with as many as possible of the existing hazardous waste management facilities to obtain any further information available. Over 300 articles were gathered and reviewed for this report.

Study Team:

This study was conducted by the Occupational and Environmental Health Program of the University of Manitoba's Department of Community Health Sciences. The team possessed considerable expertise and experience in the fields of epidemiology, toxicology, occupational

medicine and environmental health. The following individuals conducted the review and authored the report:

1. Annalee Yassi, MD, DOHS, MSC, FRCPC (Com Med), CCBOM, FRCPC (Occ Med),

   Dr. Yassi is director of the Occupational and Environmental Health Program, and Assistant Professor in the Department of Community Health Sciences in the Faculty of Medicine. She is also the director of the Department of Occupational and Environmental Medicine for the Health Sciences Centre, and is a practising occupational physician at the Manitoba Federation of Labour Occupational Health Centre. Dr. Yassi obtained her MD degree and Diploma in Occupational Health and Safety from McMaster University where she also took graduate training in toxicology. She obtained her Masters of Science in epidemiology and occupational health at the University of Toronto. She is a certificant of the Canadian Board of Occupational Medicine and has two Royal College Fellowships, one in Community Medicine, the other in Occupational Medicine. Dr. Yassi has written numerous articles and reports related to this field, and is the director of this project.

2. John Weeks, MD,CCBOM

   Dr. Weeks is Senior Advisor Health and Safety at the Whiteshell Research Institute and is currently the president of the Canadian Board of Occupational Medicine. With extensive experience in the practice of Occupational Medicine, he was one of the first Canadian physicians to become a certificant of the Canadian Board. His years of experience as an occupational physician and health advisor have earned him a well respected reputation in this field. His familiarity with assessing and communicating health risk has made him a valuable asset to this project.

3. Allen Kraut, MD, FRCPC

   Dr. Kraut recently joined the Occupational and Environmental Health Program at the University of Manitoba. After completing a fellowship in Internal Medicine at the University of Manitoba, Dr. Kraut spent three years training and practicing in the Occupational and Environmental Health Program at Mount Sinai Medical Centre in

New York City, where he had the opportunity of working with experts in the field of health effects of hazardous waste.

Summary Of Report Findings:

Overview: Despite widespread public interest in hazardous waste disposal, the science that allows determination of human health risk associated with this endeavour is fraught with uncertainties. Yet, as discussed in Chapter 1 of this report, uncertainty cannot be an excuse for inaction. Clearly failure to address the proper disposal of existing hazardous waste poses considerably greater human health risk than any well-thought through and properly executed management plan.

Routes of Exposure: Chapter 2 begins by describing the possible routes of exposure, including via contaminated ground and surface water, air, soil and food chain contamination. For a toxic substance to have an impact on health, it must be delivered in a sufficient dose by an appropriate route. People may be exposed by inhaling air contaminated with toxic substances, eating food or drinking water which has been contaminated, or directly handling toxic substances, usually either in an accidental or an occupational setting. Health effects, therefore, depend not only on the nature and quantity of chemicals disposed but on the integrity of containment. This, in turn, is a function of engineering design, the underlying hydrogeology (water and land characteristics) and meteorology (weather conditions), as well as the nature and activities of the "critical population", for example, the residential settlement patterns and related activities such as farming, fishing, hunting; the source of water supply for the surrounding community must also be considered.

Air contamination can be a major concern in the vicinity of an incinerator, either a municipal incinerator or one associated with hazardous chemicals. However, the principle route of human exposure to disposed chemicals is usually assumed to be through groundwater. Surface water and air do carry chemicals in certain instances, even in the absence of an incinerator, and it has been shown that municipal landfills can generate considerable toxic exposure. Waste that escapes into groundwater can migrate considerable distances, thus making a waste disposal issue more than of local concern. When human exposure occurs as a result of eating contaminated food, the health effects can be considerable. Appendices 2 and 3 outline studies of unmanaged

or mismanaged chemical waste disposal sites, and other environmental accidents respectively. As well, direct exposure may occur to workers employed by the facility. Therefore details are provided in this report of training requirements and health and safety program components to protect workers either employed at the facility or who may be called upon to respond to emergencies. Exposure may occur through fire and explosion, and the St. Basile-le-Grand fire has highlighted the importance to a community of this (airborne) route of exposure, again emphasizing the need for detailed protocols and safeguards. It is recognized that the risk of exposure associated with the transportation of waste from the source through to its final disposal may be considerably greater than the risk actually at facilities properly sited and designed to accept these wastes. As transportation risk assessment is the subject of a separate report it is mentioned only briefly in this study.

Possible Consequences: The full range of possible consequences of exposure are outlined in the second part of Chapter 2, noting that the health effects underlying the debate may be very subtle. While high dose levels capable of causing acute toxicity are very unlikely to be achieved even in communities where protection from exposure to toxic waste is wholly inadequate, the possibility that these exposures can exacerbate underlying conditions, or cause functional or non-specific changes must still be considered. Some studies reviewed pointed to cases in which vague symptoms and transient indicators of health effect have been suggested in association with hazardous waste disposal, but the causal connection has always been tenuous. There is considerable concern about long-term effects, particularly cancer. Possible effects on the reproductive system, neurological system, immunological system, kidney and liver are also addressed. This report addresses possible consequences of exposure in terms of the acute and chronic effects that may occur in a "worst-case scenario", and outlines these worst possible effects in more detail in Appendix 4.

Psychological Impact: The adverse psychological effects of toxic waste exposure cannot be ignored; anxiety may be real whatever its cause. The most important consideration in deciding whether or not a hazardous waste management facility should be built in a community is, in large part, the negative, or positive, psychological effect this facility may have on a community. This too is discussed briefly in Chapter 2 and addressed again in Chapter 6, which notes what experts have to say regarding the rights of a community to know, to participate and to benefit directly.

Assessing Health Risk: Chapter 3 outlines the approach to assessing health effects from hazardous waste management facilities. Hazard identification is dependent upon the studies that have been done. The highest form of evidence is from epidemiological studies, in which groups of humans are systematically studied, often over a long period of time. The success of these studies necessitates valid and accurate information regarding both the chemical exposures as well as the biological abnormality or disease that may result. Both of these are usually difficult to achieve in environmental health studies, especially in studies of chemical waste disposal sites. Moreover, even with relatively good data on exposure and target outcome of concern, often the disease in question, eg. cancer, has a long latency period, ie. it takes many years to develop. It must also be borne in mind that the outcomes are almost always non-specific, in other words, could have been caused by a multitude of factors either separately or in combination. Moreover, the statistical power to find a true difference in an exposed population is heavily dependent on the size of the community as well as how common the outcome of concern occurs in the unexposed.

Because properly designed epidemiological studies can be expensive and time consuming, much of the evidence on the toxicity of chemical exposures does not come from studies in humans. Chemicals and processes for which epidemiological and animal data show evidence of carcinogenicity (ability to cause cancer) in humans were noted. Only 13 chemicals and 5 industrial processes have been identified by the International Agency for Research in Cancer as having sufficient epidemiological evidence to be classified as definite human carcinogens. A total of only 62 chemicals have been considered as potentially carcinogenic to humans. The usefulness and limitations of other methods of determining hazards, such as animal studies, short term laboratory tests, and consideration of molecular structure are also reviewed. The evidence regarding specific chemicals likely to be of concern to a community considering establishing a hazardous waste management facility in Manitoba are discussed further in Chapter 5.

Chapter 3 also outlines the second component in risk assessment, which is exposure analysis. As noted throughout this report, there is no risk without exposure. Methods of assessing exposure, specifically environmental monitoring and mathematical modeling are discussed. The toxic substances and the possible health effects are determined, and once the extent of exposure has been characterized, the next step is to estimate possible human response to that exposure. For some toxic substances, a threshold is assumed to exist, in other words a certain level of exposure below which health effects are unlikely to occur. For other substances,

including all potential carcinogens, the prudent approach which is generally accepted is to assume that no thresholds exist, although the magnitude of the risk at low level exposure is highly contentious.

The final step in risk assessment, addressing the question of "how much harm is probable" is to combine the above information with specific information on possible exposures to be encountered in the actual hazardous waste management facility in question. This requires integrating information from this report with that obtained from projections of the waste streams and other relevant data.

Studies to Date: Chapter 4 reviews the numerous studies that have been conducted to date on unmanaged or mismanaged waste disposal sites as well as "managed" hazardous waste disposal sites. To paraphrase a noted expert in this area: Although studies do not confirm a causal association between exposure to hazardous waste sites and health effects, they cannot be interpreted to indicate that no risk exists, but rather that no serious health effects clearly attributable to a specific chemical or groups of chemicals have been identified to date by all the study methods employed. While the vast majority of studies regarding unmanaged or mismanaged chemical waste disposal sites show either no effect or are equivocal, as outlined in Appendix 2, some findings that have raised concern should be mentioned. The evidence from Love Canal insofar as it relates to low birth weights and reduced stature is troublesome. The elevated incidence of leukemia at Woburn initially reported seems to be supported by more recent studies, and despite the fact that the evidence of the relationship to hazardous waste can not be considered causal, it is persuasive. The role of specific chemicals encountered in a chemical waste disposal facility in Clinton, New Jersey in the causation of bladder cancer is consistent with what was previously known about these substances and therefore is also noteworthy. These and similar events point in the direction of a link between adverse health effects and exposure to contamination from ill-managed toxic waste disposal areas, although should still be interpreted with caution.

Despite considerable literature searching and personal communications, few reports concerning adverse health effects around managed waste disposal sites were found. This may be because the job has been well done and there are no effects to detect, or possibly the fact that engineered waste management is a comparative recent phenomenon such that long term adverse effects have not yet had time to become apparent, and therefore have not been sought. In the

literature reviewed and personal contact with two of the well designed hazardous waste management facilities in existence, there were no concerns articulated regarding health effects. The reports that were found of adverse health effects from managed waste disposal sites related mainly to incinerator emissions, with the exception of the Stringfellow report which noted short-term health effects possibly attributable to overflows in the containment pond.

Perspective: The hazards discussed in this report must be kept in perspective. To assist in providing an overall perspective of health risks, this report contains a table which outlines risks from various activities of daily life. However, it cannot be adequately stressed that voluntary risks should not be compared with involuntary risks, as even when the latter may be tiny in comparison to the former, these may still be unacceptable.

Recently another well respected environmental health authority wrote: "In the nineteen years that I have been answering the telephone, there have been very few instances where I felt that there was compelling evidence that the health of people was being compromised by community exposure to toxic wastes..... I find little evidence to support the view that the public's health is compromised by most of the toxic related episodes which receive media attention." (Milham, 1988). It is noteworthy that workers' compensation boards have recognized as compensable only a relatively small number of cases of disease occurring in occupationally exposed individuals. As occupational exposures are almost always higher than even worst case continuous environmental exposures, actual disease in the general population from environmental exposures is still often in question, except in a few serious mishaps, as occurred in Bhopal, India, or Minimata, Japan for example.

Summary of Conclusions:

After reviewing well over 300 articles and documents, it is concluded that the evidence of adverse health effects attributable to hazardous waste disposal sites is equivocal. Much of the information is lacking in statistical significance, some is inferential and some indicates that no adverse effects are detectable. It is concluded that in all likelihood some short term adverse health effects can indeed be attributable to mismanagement of toxic waste, and that in a small number of other incidents possibility exists that more serious health effects have indeed occurred. While the information related to health effects possibly attributable to unmanaged or

inadequately managed hazardous waste is equivocal, there is virtually no evidence of health effects attributable to the operation of waste management sites where appropriate current technology is in use. Thus, while the health risk of a hazardous waste management facility is not zero, it is not thought to be high in comparison with risks of everyday acceptable activities of life.

As such, the conclusion of this report is that we are not able to define any risk associated with a well-managed facility. Those individuals who expected this report to provide a definitive answer to the question: "Will a properly engineered hazardous waste management facility located in their community be safe?" may therefore be disappointed. However, safety is a relative concept. This report provides a perspective in which this issue could be evaluated.

Summary of Recommendations:

As the first theme that characterizes this report is the theme of uncertainty, it is recommended that computerized data banks are established to carefully monitor the waste stream; a well designed strategy of comprehensive environmental monitoring is also essential. While biological and health effect monitoring should not be seen as protective to a community in any way, such endeavours may aid future attempts to assess health risks. It is recommended that consideration be given to establishing a baseline health assessment, with the details of this to be explored in consultation with the medical community of the region in consideration.

The second theme of the report relates to the importance of a community receiving full access not only to all information, but to participation in all decision-making bodies. While this point does not relate directly to the medical literature, it is felt that many of the perceived health problems reported in the past could have been avoided if more attention had been paid to measures to mitigate the psychosocial impact of a hazardous waste disposal facility.

CHAPTER 1

INTRODUCTION

Almost daily, television and newspapers show pictures of citizens demonstrating their concern about toxic substances. There is general agreement that "toxic waste" can indeed be hazardous to human health. Perhaps more importantly though, there is also general consensus that a system must be found to manage properly these substances so that health risk is reduced to as low a level as feasible. The purpose of this report is to discuss health effects of hazardous waste and to emphasize human health concerns related to establishing a hazardous waste management facility. As explained in the glossary, "hazardous waste" includes various sorts of substances which may pose a hazard. In that public concern generally focuses upon those substances which are potentially hazardous by virtue of their toxicity, this will be the focus of this report as well.

As modern society has become increasingly industrialized over the past century, the proportion of health risks arising from human activity and specifically from industrialized and technologically advanced society, has grown and changed dramatically. Initially, the control of infectious diseases was paramount. With urbanization and increasing residential crowding, the potential for food and waterborne infectious epidemics underlined a need to deal with human sanitary waste. As microbiological knowledge advanced, most infectious disease problems have come under control, aided in large part by the proper management of human waste, such as proper sewage facilities. Now, health risks associated with chemical or radiation exposure have commanded increasing attention. Non-infectious disease hazards were originally recognized in occupational settings where exposures were particularly intense and sustained. More recently, concern has turned towards potential toxic exposures in all phases of human activity, occupational as well as non-occupational. The shift of concern from microbiological to chemical exposures has also been accompanied by a shift in concern from the acute illnesses arising from relatively high levels of exposures to the long-term or chronic health effects resulting from lower levels of exposures.

Unfortunately, the methodology needed to establish risk assessment associated with these low levels of exposures is fraught with difficulties. The effects often have long latency periods,

(in other words, many years between the exposure and the onset of disease,) and the results of studies of these effects are often ambiguous. The medical and scientific communities have, for the most part, been unable to provide complete answers to questions raised by society regarding potential health effects of environmental exposures. These limitations will be discussed in this report.

It is important to underline, however, that uncertainty is no excuse for inaction. There are large numbers of chemical waste "disposal sites" in Canada. These range in quality from piles of unrecorded corroding drums and forgotten landfill sites now covered over, to municipal "dumps" or sewer systems, to the few carefully constructed engineered facilities, with liners, fences, and monitoring. While the Love Canal incident and numerous other highly publicized toxic waste disposal incidents outside of Canada became almost an obsession with the media, there is still remarkably little consensus on the actual extent of hazard created by improper hazardous waste disposal. Nonetheless, what is abundantly clear is that the failure to address the disposal of toxic substances provides far greater hazards to human health than any effects that could possibly be attributable to a properly sited and designed hazardous waste management facility in the community.

The issue however, is the definition of community. Is one's community simply one's family? Does it extend to the several blocks surrounding one's household? the neighborhood? the town? or some larger region, province, country... or even planet, of which one is an inhabitant? To some extent, we all use the products which generate the hazardous waste. Yet even if a community considers it a social duty to play a role in promoting the safest possible management of hazardous waste, must a community trade off its own health for assuming this social responsibility and the economic advantages that this may bring? Certainly the answer must be "no". The purpose of this report, therefore, is to provide ample information so that a community may begin to examine this question, and decide for itself if the magnitude of the health risk associated with hosting a waste management facility is acceptable.

CHAPTER 2

HOW HAZARDOUS WASTE COULD CAUSE HARM

2.1 - POSSIBLE ROUTES OF EXPOSURE

As noted above, human exposure to toxic waste can occur through ground and surface water, air, soil, food and direct contact. Numerous factors influence the extent to which a given chemical is likely to result in human exposure. These are discussed at length in many articles, symposiums and textbooks including Grisham (1986) and Andelman and Underhill (1987). Chemical characteristics affect deposition and interaction with other substances and organisms; physical characteristics such as solubility and volatility influence the rate at which a chemical leaches into groundwater or escapes into air; existing environmental and meteorological conditions influence exposure, and, of course, the method and integrity of containment is of paramount importance. With respect to chemical and physical properties, the greater the solubility in water, the more likely a chemical will leach and migrate in groundwater; for low solubility compounds, high density (heavy) substances will sink whereas low density substances will float near the surface; the higher the vapour pressure, the more likely the compound will evaporate from water or soil into air. If a compound is not readily biodegraded (such as PCBs) it will be persistent. With respect to environmental factors, the presence in the atmosphere of suspended dust will increase exposure as some chemicals could adsorb onto the particles; and the nature of the soil is a key determinant of the likelihood of water contamination. Clays or organic rich soils absorb many chemicals, thus reducing the rate of release; in contrast, sand and gravel facilitate rapid movement of water and other liquids and do not provide good containment. Microorganisms, numerous in organic rich soils, can interact with chemicals changing their nature to either more or less toxic substances (Freed et al., 1977). Numerous texts, articles, seminars and conferences are devoted to exploring these interactions. Indeed the crucial question of the likelihood of exposure under particular circumstances merits intensive study.

Before exploring each of these exposure circumstances in greater depth it should be noted that the risk of a transportation mishap is always present. These could occur anywhere from the source of the waste to its final disposal site. There is nothing unique to hazardous waste in this regard, as spills of agricultural products, solvents, petrochemicals and other toxic substances occur all too often. The risk of transportation accident must be considered in siting a facility,

but will not be extensively discussed here, as it is the subject of a separate report (Dillon, 1989).

2.1.1 Air Contamination: When an incinerator is present, the potential for air contamination is evident (cf. Travis and Cook, 1989). However, even when there is no smoke stack to emit substances into the atmosphere, the air surrounding a waste disposal site may become contaminated from a variety of other sources. Evaporation from uncovered surface impoundments could occur; hazardous waste from landfills could be transported upwards as gases through the soil and into the atmosphere (Shen, 1981; Thibodeaux, 1981; Grisham, 1986), although this may take months or years depending on the substance and the disposal method; fugitive emissions may occur from processing operations. Minott (1989) recently compared the health risks from stack emissions at energy recovery facilities with that from municipal landfills. While an energy recovery facility is a type of solid waste or garbage incinerator not a hazardous waste incinerator, Minott's observation is interesting. He noted that landfill gas chiefly comprises the explosive gas, methane, but also contains odorous sulphur compounds and volatile organic compounds, some of which being known carcinogens (substances that could cause cancer). He contends that municipal landfills are more dangerous than resource recovery facilities, noting that some carcinogens have been measured in levels exceeding ambient standards at municipal landfills in California, Wisconsin and New Jersey. Again, scientific evaluation has developed to a sophisticated level to predict and control such exposures, as Minott (1989) also describes.

Not only is the burning of open landfill a hazardous practice, but burning municipal garbage in incinerators is also hazardous. The National Incinerator Testing and Evaluation Program (NITEP) of Environment Canada have documented a myriad of toxic chemicals from municipal incinerators (Mohr, 1988). As Mohr (1988) also notes, new anti-pollution technologies, such as scrubbers, can reduce stack emissions of toxins to barely detectable levels. These and other sophisticated devices would be expected to be employed in any hazardous waste incinerator.

2.1.2 Water Contamination: Whereas contamination of air tends to be episodic and of short duration, since it usually depends on precipitation, temperature and wind, groundwater contamination may be more problematic as it usually moves slowly with little mixing and

dilution. Also, contaminated groundwater moves in a plume that may persist for decades after polluting activities have ended. It must be kept in mind that most "chemical disposal sites", as discussed in the media as well as in scientific journals, refer largely to uncontrolled sites, including open dumps, landfills, bulk storage containers, and surface impoundments. The locations of these sites were usually selected for convenience and expedience with little regard for even the type of soil in which they were located. Some were located only a few feet above the groundwater, frequently in close proximity to shallow drinking water wells. When the sites were abandoned, they were frequently left uncovered, thus increasing the likelihood of water pollution from leaching or flooding, as well as increasing the chances of air pollution and direct contact.

The diversity of chemicals reported in drinking water aquifers in the vicinity of such chemical disposal sites include chlorinated solvents, aromatic hydrocarbons, pesticides, trace metals, and polychlorinated biphenyls. The chemicals penetrate gradually into the groundwater zone from points of surface or subsurface disposal, are transported very slowly, and may resist biological and chemical degradation so that they persist for an extremely long time.

The well developed science that is used to predict the behavior of contaminants in groundwater, namely hydrogeology, was not applied in the situations discussed below. Also, it must also be recognized that waste water is not the only source of groundwater contamination. Other industry-related sources include leaks of chemicals from storage areas and accidental spills. Automobile service and repair shops, dry cleaning, printing and pesticide operations are common sources of water contamination. Road run-off, municipal landfills, and domestic waste water are also important sources. As noted by Miller (1981), household products contain many soluble organic chemicals that find their way to septic tanks, from where they migrate to the water table. Some products used for cleaning septic tanks, in fact, contain suspected carcinogens. Ironically perhaps, the chlorination of drinking water is the subject of current debate, as there is some evidence to suggest that this process generates carcinogenic compounds, now implicated in an increased risk of several cancer types (Wilkins et al, 1979; Shy, 1985).

Loss of a groundwater source due to contamination can be a severe economic hardship on the community or the homeowner. Hydrogeological surveys at chemical disposal sites are described in detail by Miller (1981), Ragone (1987) and others. Exposures resulting from water

contamination both from waste sites as well as other sources and the consequences of these exposures are discussed further in Chapters 4 and 5.

2.1.3 Soil and Food Contamination: Depending on the factors cited above, hazardous waste can adsorb onto soil particles, which may result in human exposure via direct contact with the soil, inhalation of particles transported via wind, erosion, or exposures to suspended particles in surface waters, (Grisham, 1986). Furthermore, hazardous waste may be lost from the soil via uptake by plants and animals living on or near the soil (Litwin et al, 1983 as cited by Grisham, 1986). Animals may then be further exposed by consuming contaminated plants or other animals. Human exposure could occur by eating contaminated food crops, contaminated wild game or grazing farm animals which contain bioaccumulated hazardous chemicals (i.e. chemicals which have become concentrated up the food chain). Eating contaminated fish could be a significant source of contamination, if fish comprises a large part of the diet. For example, human consumption of DDT-contaminated fish from Indian Creek near Triana, Alabama, resulted in high blood levels of this chemical (Kreiss et al, 1981).

It is also important to note that soil contamination occurs through other means than dump sites. One example of an industrial discharge related soil problem, as discussed by Olson et al (1988), is the use of dioxin contaminated sludge from papermills, which, at least until recently, has been applied to agricultural lands to enhance soil.

Scientists have been able to develop methods to predict the sorption potential of chemicals, uptake by plants of various sorts, and bioconcentration factors, so that these factors could be taken into consideration in the siting and design of facilities for the management of hazardous wastes. Again, this science was either unavailable or does not appear to have been applied in the many "chemical waste dumps" described.

Appendix 2 summarizes the methods and findings in a variety of studies of chemical waste disposal sites; these and others are discussed further in Chapter 4. Appendix 3 summarizes incidents of accidental releases of chemicals into the environment; Chapter 5 discusses health effects of some of the exposures involved along with others of potential relevance in a Manitoba hazardous waste management facility. While evidence regarding health effects in populations exposed from waste disposal facilities may be equivocal, accidental

releases of chemicals into the environment (eg. as occurred in Bhopal or Minimata and the numerous other examples cited in Appendix 3) provide ample evidence that serious health effects can indeed occur if contamination is severe. In situations in which food supplies were contaminated, sequelae were often rapid and severe, with skin, gastrointestinal, and central nervous system effects predominating. Contamination of water supplies, which are presumably of much lower concentrations, did not usually result in clear cut responses, even in more blatant situations of exposure. In situations in which toxic chemicals were not identified in food or water, unequivocal health effects have rarely been detected. This highlights the importance of locating hazardous waste management facilities in the appropriate hydrogeological locations, properly designing and engineering the facility, and top notch management of the process. As discussed by Schweitzer and Williamson (1987) new techniques have been developed to monitor water and soil, and indeed U.S. regulations require certain types of groundwater monitoring. Any newly built hazardous waste management facility would certainly be expected to apply state-of-the-art control technology.

2.1.4 Occupational Exposure

Arguably the people at greatest risk of exposure to toxic chemicals are the people employed in the facilities where these chemicals are manufactured, utilized in other production, stored, transported, and disposed. The U.S. Occupational Safety and Health Administration (OSHA) has issued a final standard specifying measures to protect workers in hazardous waste operations. The standard covers workers employed in clean-up operations at uncontrolled hazardous waste sites as well as facilities that are licensed for waste transport, storage and disposal, as well as workers responding to emergencies involving hazardous materials, including police, ambulance workers, and fire fighters. (A detailed description of the Occupational Safety and Health Requirements for these workers is provided in a booklet recently released by OSHA, 1989). A written safety and health program is required that identifies, evaluates and controls occupational health and safety hazards, and outlines emergency response procedures for hazardous waste operations. Detailed requirements are provided on the following topics:

1) An organizational work plan, including protocols outlining supervisory and employee responsibilities and means of communication in various situations;

2) Site evaluation and control, including initial and periodic surveillance activities;

3) A site-specific program, which outlines the safety and health measures specific to those characteristics unique to the particular site;

4) Information and training program as outlined in Table 1;

5) Personal protective equipment program, which includes an explanation of equipment selection and use, maintenance and storage, decontamination and disposal, training and proper fit, in-use monitoring, program evaluation and equipment limitations;

6) Monitoring, including air monitoring and specific monitoring as indicated;

7) Medical surveillance program, including preplacement, periodic, and termination of employment medical assessment;

8) Decontamination procedures, outlining, for example, how contaminated clothing should be handled; and an

9) Emergency response program, detailing plans to cover all eventualities, including evacuation routes and procedures if the need arises.

Engineering controls and work practices are deemed preferable to personal protective equipment. An example of a suitable engineering control is the use of pressurized cabs or control booths on equipment; examples of safe work practices are the removing of all non-essential employees from potential exposure while opening drums, wetting down dusty operations, and placing employees upwind to potential hazards. In Canada, the Workplace Hazardous Materials Information System (WHMIS) outlines particular requirements for the training of workers exposed to toxic chemicals and other control products. It also outlines the necessity for labelling and Material Safety Data Sheets. Moreover, the of Dangerous Goods Handling and Transportation Act has further requirements to ensure that the chemicals that arrive at a site are properly identified. It would be expected that a community advisory committee to a hazardous waste management facility would become familiar with this and other relevant legislation.

2.1.5 Transportation Risk

While the focus of this study is related to health concerns associated with a hazardous waste management facility, it must be appreciated that perhaps the greatest potential for exposure occurs not at the central treatment and disposal site itself but along the transportation route. Typically hazardous wastes are either liquid wastes and transported in tanker trucks, or are non-pumpable and transported in van trucks. There may be transfer stations such that all hazardous waste need not be shipped directly to the central treatment and disposal facility, thereby decreasing the number of hazardous waste long-distance transports required. Dillon (1989) has found that 11 percent of the trucking traffic in Manitoba currently carries dangerous goods; this is consistent with the 10 to 13 percent found in U.S. surveys. These include carriers of fuels, liquefied gases, poisonous or corrosive substances and miscellaneous others. Thus risks associated with transportation of hazardous waste should be analyzed as an incremental risk in hazardous substance movement. Dillon (1989) has developed a method of transportation risk assessment that can quantify risk associated with a proposed transportation system, both in absolute terms and in comparison to current risk associated with comparable dangerous goods traffic. This information is obviously crucial in siting decisions regarding hazardous waste management facilities.

TABLE 1

TRAINING REQUIREMENTS OF THE U.S. OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA 1989) FOR HAZARDOUS WASTE FACILITY WORKERS

HAZARDOUS WASTE CLEAN-UP SITES	
STAFF	
- Routine site employees	40 hours initial 24 hours field 8 hours annual refresher
- Routine site employees (minimal exposure)	24 hours initial 8 hours field 8 hours annual refresher
- Non-routine site employees	24 hours initial 8 hours field 8 hours annual refresher
SUPERVISOR/MANAGER OF	
- Routine site employees	40 hours initial 24 hours field 8 hours hazardous waste management 8 hours annaul refresher
- Routine site employees (minimal exposure)	24 hours initial 8 hours field 8 hours hazardous waste management 8 hours annual refresher
- Non-routine site employees	24 hours initial 8 hours field 8 hours hazardous waste management 8 hours annual refresher
TREATMENT, STORAGE AND DISPOSAL SITES	
STAFF	
- General site employees	24 hours initial or equivalent 8 hours annual refresher
- Emergency response personnel	Trained to a level of competency Annual refresher
OTHER EMERGENCY RESPONSE STAFF	
Level 1 - First responder (awareness) level[1]	Sufficient training or proven experience in specific competencies Annual refresher
Level 2 - First responder (operations level)[2]	Level 1 competency and 8 hours initial or proven experience in specific competencies
Level 3 - HAZMAT technician[3]	24 hours of Level 2 and proven experience in specific competencies
Level 4 - HAZAMAT specialist[4]	24 hours of Level 3 and proven experience in specific competencies
Level 5 - On-the-scene incident commander[5]	24 hours of Level 1 and additional competencies Annual refresher

1 Witnesses discovers a release of hazardous materials and who are trained to notify the proper authorities.

2 Responds to releases of hazardous substances in a defensive manner, without trying to stop the releases.

3 Responds aggressively to stop the release of hazardous substances.

4 Assumes control of the incident scene beyond the first-responder awareness level.

2.2 POSSIBLE CONSEQUENCES OF EXPOSURE

The types of health effects which may be linked to toxic substances depend, of course, on the nature of the substances as well as the route, dose and time course of exposure. Illustrated by the studies outlined in Appendix 2 and discussed in Chapter 4, none of the dozens of public health investigations have to date produced an uncontestable link between chemical waste exposure even in the most uncontrolled situation, and serious adverse health effects. On the other hand, they have also failed to establish the benign nature of these disposed toxic chemical wastes. Limitations of the study methods are discussed further in Chapter 3. The purpose of this section is to outline the range of possible effects in a "worst case scenario", noting only briefly some of the problems associated with studying these effects.

2.2.1 Acute Symptoms and Range of Effects: Acute symptoms, or effects that are noticeable soon after exposure, are usually short lived, and generally occur at dose levels associated with accidents or occupational exposures. For example, an individual acutely exposed to high levels of solvents, will be expected to experience headache, lightheadedness, nausea, and a variety of other central nervous system symptoms, and possibly numbness, tingling and symptoms of other peripheral nervous effects, as well as symptoms due to effects on other organ systems, depending on the nature of the substance. People acutely exposed to high levels of dust, will, of course, experience respiratory tract symptoms, including possibly chest discomfort, cough, wheeze and shortness of breath.

Conditions and symptoms associated with specific exposures of particular concern (i.e. pesticides, polychlorinated hydrocarbons and certain metals) are discussed further in Chapter 5, and are outlined broadly in Appendix 3. Buffler et al, (1985) prepared a series of tables outlining the adverse health effects reported to be due to exposure to chemicals, according to target organ affected. These tables, summarizing possible effects on a) skin, b) nervous system, c) liver, d) reproductive system and fetus, e) hematopoietic (blood forming) and lymphatic system, f) genitolurinary system, g) respiratory tract, h) gastrointestinal tract, and i) cardiovascular system, are presented in an adapted form in Appendix 4. It should be kept in mind though, that these effects are known not from toxic waste exposure but exposure to the actual chemical in often very high dose exposure situations. Nonetheless, they are relevant to appreciate in a "worst-case scenario" appraisal.

2.2.2 Aggravation of Health Problems and Non-specific Effects: Serious acute symptoms are unexpected with low levels of exposure. However, levels of exposure not out of keeping with what could be associated with environmental exposures can be associated with exacerbation of underlying conditions, and may be linked to non-specific mild symptoms of uncertain significance. Exposure to sulphur dioxide and particulates, associated with air pollution attributable to a variety of industrial sources, are known to be associated with an aggravation of asthma, increased respiratory tract infections, and increased hospital visits particularly in children with chronic respiratory conditions (Linns et al, 1983; McDonnel et al, 1985; Bates and Sizto, 1983; Bates and Sizto, 1989). The relationship between air pollution and breathing problems is well established.

A recently published study (Ozonoff, 1987) focused specifically on acute symptoms in a neighborhood contaminated by hazardous waste. In this situation a five acre site was located less than 400 feet from a residential neighborhood in Lowell Massachusetts. About 250,000 gallons of chemicals were received monthly by this commercial enterprise. After the first few years of operation financial problems led to significant breaches of acceptable waste management practices at the facility, causing neighborhood residents to complain of strong chemical odours. Some residents also reported various health problems that they associated with periods of increased odours (Ozonoff et al, 1987). When the operations finally ceased in 1977 because of bankruptcy, it was found that there had been repeated spills with leaking onto the ground. By 1982, five years after cessation of the operation, residents continued to complained of odours and health effects. Air monitoring was conducted for thirteen priority chemicals; of these, benzene, toluene, trichloroethylene and tetrachloroethylene were detected in the air in the surrounding residential neighborhood, although at levels below the odour thresholds for these ubiquitous chemicals and nowhere exceeding 20 parts per billion (ppb), usually measuring 10 ppb (Ozonoff, 1987). Nonetheless a symptom prevalence survey of neighborhood residents was conducted with a comparison group consisting of a population further from the site. The study demonstrated that residents living near the source of airborne chemical contaminants reported more respiratory symptoms (wheezing, cough, persistent colds), more sensations of irregular heart beat and medical history of heart trouble, more constitutional complaints (fatigue, lassitude, bowel dysfunction), and a more frequent medical history of anemia and other blood disorders, compared with the comparison population. Reports of eye and nose irritation were also

associated with proximity to the site. The differences could not be explained by differences in age, gender, occupational experiences, or various lifestyle characteristics.

However, as the authors (Ozonoff, 1987) and numerous others (eg. Sackett, 1979; Roht et al., 1985; Marsh and Caplan, 1987; Hopwood and Guidotti, 1988) explain, a study such as this has a serious problem of "recall bias", (ie. differential recall if exposed versus non-exposed, a problem discussed further in Section 3.1.1). Several aspects of this study do point to recall bias being significant. For example, there was a dramatic "pan symptom" effect, i.e. an answering of "yes" to a vast array of symptoms some of which are highly unlikely to be attributable to the exposure substances.

Nevertheless, perhaps more important than the reasons for these symptoms, the perception of ill health is an objective fact on its own of some significance. The perception of symptoms, whether or not there is a documented physiological basis, has an adverse impact on the quality of life. As such, a higher prevalence of respiratory symptoms and headaches are important, even though relevant occupational health literature indicates that these symptoms are thought only to occur after exposures that are very much higher than those measured by the concurrent air monitoring - in some cases three orders of magnitude higher. Because occupational groups have to be at least healthy enough to be employed, and often those who are particularly sensitive to these effects leave the workplace early, it is possible that respiratory symptoms and headaches can indeed be attributable to very low dose exposures that can come from poorly managed waste facilities, particularly in those who were predisposed to such symptoms.

Exacerbation of pre-existing conditions, such as bronchitis or emphysema, and non-specific deterioration of health or sense of wellbeing were also noted by Harris and Highland (1984) studying a Tennessee hazardous waste disposal site. Other reports of non-specific symptoms in areas of chemical disposal sites are noted in Appendix 2.

2.2.3 Laboratory Evidence of Organ Dysfunction: Another type of effect, which may or may not be accompanied by symptoms, are effects which can be detected in laboratory tests related to liver, kidney, neurological, or immunological function. The clinical significance of these changes is often uncertain. A variety of screening tests exist to detect subtle changes in these organ systems which can, and have been, used to assess effects of low level exposure. For example, one study found transitory changes in liver function associated with exposure to a site containing

carbon tetrachloride, hexacyclopentadiene, and hexachlorocycloheptadiene (Clark et al., 1982). Another example is changes in liver function associated with PCB exposure.

The laboratory tests indicative of liver and kidney damage, along with some examples of environmental toxins that have been shown to be able to cause these changes are discussed extensively by Grisham (1986). It must be recognized that there is a wide range of values for laboratory tests in healthy populations, with "abnormal" values more often than not overlapping "normal" values. Because of the problem of "false positives" and "false negatives" in laboratory tests, interpretation of results must always be based on consideration of other clinical factors.

2.2.4 Reproductive Problems: Reproductive problems have been the focus of particular concern in investigation of possible hazardous waste exposures. These problems, including impotence, sterility, fetal loss, stillbirth, low birth weight, birth defects, and infant illness and death, have been targeted with respect to hazardous waste exposure for pragmatic as well as biological plausibility reasons. The June 1981 Rockefeller University Symposium concluded that reproductive dysfunctions are the most promising endpoints for epidemiological investigations of waste site exposed populations (Lowrance, 1981). From a pragmatic point of view, some of these data are already collected. For example, data on perinatal mortality, birth weight and sex of liveborne infants are routinely recorded in most jurisdictions. In some jurisdictions registries are now being developed for congenital malformations. For other reproductive outcome, however, such as spontaneous abortion, or changes in semen, special studies need to be mounted. The advantages and disadvantages of focusing on specific aspects of reproductive function are discussed at length by Stein et al., (1981), Warburton (1981), Marsh and Caplan (1987) and others.

Table 2 outlines some of the negative reproductive outcome that could be studied, and their frequency in the population. Impotence, sterility and infertility are generally not easily studied since they are difficult endpoints to measure accurately, and there are many confounding variables. Semen abnormalities can be analyzed as indicators of exposure, however their significance for reproductive function is largely unknown. Spontaneous abortion, defined as termination of pregnancy before 28 weeks of gestation, may be a good indicator of reproductive dysfunction and exposure to toxic substances. Normally, spontaneous abortion acts to screen out about 90% of all chromosomally abnormal conceptions and about 50% of all

malformed fetuses. At least 15% of all recognized pregnancies are spontaneously lost, and the number in unrecognized pregnancies may be much higher.

The higher the rate in the background population, the greater is the power to detect a change, given a limited population size, as discussed further in Chapter 3. A doubling in the rate of spontaneous abortions would not be reliably detected unless there had been 118 pregnancies in that population (Stein et al., 1981) Another, disadvantages in studying spontaneous abortions is the fact that the only sources of available data are medical records and personal interviews. Medical records are insufficient because a large number of spontaneous abortions are not brought to medical attention, and interview data are subject to recall bias and inaccuracies due to self-diagnosis.

Congenital malformations or birth defects are much less common than spontaneous abortion, occurring in only approximately 2% of live births, as shown in Tables 2, although at least the more visible of these malformations are likely to be reported within the community. Over 1000 live births are necessary in a population to reliably detect a doubling in birth defects (Stein et al., 1981). Another problem is that these malformations may be caused by a variety of agents, including maternal drug ingestion during pregnancy, viruses, and a wide variety of environmental exposures. The new registries that are being developed will help facilitate analysis of birth defect patterns in the future.

Low birth weight is a useful indicator of maternal health and is strongly associated with neonatal death, congenital malformations, developmental delay, and chromosomal abnormalities. Also, as noted above, birth weights are routinely collected on birth certificates and hospital records. This measure also has considerable power in statistical analysis, due to relatively high frequency and because it is a continuous variable. For these reasons, as noted in Chapter 4, the reported increase in low birth weight infants is amongst the most convincing of all health effects described from the Love Canal (Vianna and Polan, 1984; Goldman et al., 1985; Paigan and Goldman, 1987) Confounders that must be taken into consideration however, include maternal age, parity, smoking habits, and gestational age (Warburton, 1981).

An understanding of the limitations of these measures is crucial to analysis of studies on health effects in hazardous waste exposure. The less common the event, the larger numbers are necessary in a study to ascertain if an increase in the event has indeed occurred. Quite apart

from the need to control confounding variables, as listed above, it is clear why determining adverse effects on reproductive health is not easy in a small community.

2.2.5 Immunological effects: The immune system is a delicately balanced system of many different cell types in the body. An imbalance in this tightly regulated network, which may result from cellular damage caused by infections, drugs, stress, and various toxic chemicals, can have a variety of effects. Immune alterations may result in immune enhancement, which might ultimately result in autoimmunity, contact hypersensitivity or allergy, or it may cause immune suppression which might result in decreased resistance to bacterial, viral, fungal, and parasitic diseases, as well as an increased incidence of neoplastic diseases such as cancer caused by the impaired immune surveillance.

Appendix 5 provides a partial list of chemicals and combination of chemicals found in various animal species to cause effects on the immune system ; the type of immune dysfunction and animal species in which the results were found are listed as well (c.f. Grisham, 1986). Spreafisco and Vecchi (1984), suggested that an immunotoxic substance can cause effects at doses lower than those producing general signs of toxicity in other body systems. Studies of accidentally exposed human populations in Michigan, Japan, and China suggest that polybrominated biphenyls (PBBs) and polychlorinated biphenyls (PCBs) may cause temporary immune dysfunction in humans exposed to sufficient concentrations of these chemicals (Bekesi and Selekov, 1978). There is no convincing evidence of long-term effects on the immune system in these or other population studies. Much research is needed in this area.

2.2.6 Neurotoxic Effects: There are numerous chemicals known to cause severe effects on the neurological system. Indeed, many of the most publicized effects of toxic exposures have resulted in damage to the nervous system. Neurological poisoning occurred in the population surrounding Minimata Bay exposed to mercury effluent dumped into the bay (Tedeschi, 1982). Numerous examples of neurotoxic injury from alkane hydrocarbons have been cited in the occupational and environmental health literature (c.f. Grisham, 1986), although, again it is difficult to determine whether a chemical or mixture of chemicals at a chemical disposal site presents an actual neurotoxic danger to the public (Schaumburg et al, 1981, 1983).

A partial list of chemicals and their neurological effects are presented as Table 3. There are a number of confounding variables that must be considered in evaluating chemicals for potential neurotoxicity, including the many clinical syndromes that may effect symptoms, such as diabetes melitus or other pre-existing conditions in the population. As illustrated in Table b of Appendix 4, these changes may be subtle, such as disturbances in visual acuity, memory, problem solving ability and attention span as well as irritability and mood alterations. While no firm conclusion can be drawn as to the effect of a chemical waste disposal site, these possible subtle effects on the nervous system must be considered as plausible in a "worst-case" scenario.

2.2.7 Cancer: The chronic effect of greatest concern in society today is that of cancer. It is known that certain substances are indeed capable of inducing cancer. Evidence of carcinogenicity in humans and animals, as determined by the International Agency for Research on Cancer (IARC, 1982) is shown in Table 4. However, there has not been a single study of a community exposed to hazardous waste that has shown an unequivocally increased cancer risk. The situation of Woburn, discussed further in Chapter 4, is the only one in which there has been a serious contention of an increase in cancer caused by toxic waste exposure, in this case in childhood leukemia (Lagakos, 1984); even this has been refuted (Grisham, 1986).

Milham (1988), noting the problems in extrapolating to the community from the workplace, and other methodological limitations, nonetheless states: "My operational rule of thumb is that if an occupational cohort with very heavy chemical exposure does not show a health problem then community exposures will probably not show any health effect". The limitation of occupational epidemiological studies notwithstanding, Milham believes that the occupational literature should be quite reassuring to communities exposed to toxic waste. Chapter 5 discusses cancer risk associated with specific chemicals, noting, for example, that even with respect to PCB, despite what the media would have us believe, evidence of a serious cancer risk to humans is extremely limited.

2.2.8 Psychological Effects: Although there is still very little evidence that chemicals from dump sites have ever truly made people sick, there is convincing evidence that exposures from these sites have affected the psychological well being of communities. As concluded by Holden (1980) in referring to the Love Canal incident: "The psychological damage from this long running disaster may ultimately turn out to rival and perhaps exceed physical damage."

Although there is considerable uncertainty with respect to the physical effects, there is no denying that events such as the Love Canal have not promoted family unity. Among the 237 families involved in the 1978 evacuation of Love Canal, 40% of the couples separated or divorced, slightly higher than the usual 30-35%. The author states that typically the wife wants to leave and the husband says they cannot because he has invested his life in the home and his work. So the wife leaves with the children. This is debilitating to the family and particularly to the man whose image as the protector of his family is thrown into chaos. The Niagara Falls Community Mental Health Centre attempted to relieve distress by deploying outreach workers and conducting "coping with stress" sessions. However, perhaps largely due to the stigma of mental disorders, particularly among a community who ascribed their problems to either chemical or other causes, there were very low turnouts to these sessions (Holden, 1980).

Susser (1985) documents how health concerns of residents, industrial workers, and plant management cannot be interpreted without considering problems of unemployment, political affiliation, as well as company policies and their impact over time. She describes how the willingness of a community to accept exposure is largely attributable to how it sees the benefits associated with this exposure. Sense of well-being and health behaviour thereby becomes very much linked to the degree of trust a community has in the agency or organization responsible for the exposure and its control, and other socioeconomic and psychological factors which they associate with this situation. Impending litigation is a very important confounding factor in this regard. Stress generated by uncertainty associated with hazardous waste facilities and how to cope with, mitigate, or eliminate this stress are discussed further in Chapter 6.

TABLE 2

SELECTED INDICES OF REPRODUCTIVE DYSFUNCTION,
AND THEIR FREQUENCY,
IN THE POPULATION*.

INDEX	FREQUENCY
Sexual Dysfunction	
decreased libido	Not Measurable
impotence	
Sperm Abnormalities	
abnormal shape	
decreased speed	
decreased number	
absence of sperm	1/100 men
Decreased Fertility	
abnormal genital development	
amenorrhea	
anovulatory cycles	
infertility	10-15/100 couples
Sponteneous Abortion (<28 weeks)	~15/100 pregnancies
Perinatal Death	
Stillbirth (>28 weeks)	2-4/100 births
death in first week	
Decreased Birthweight	
<2500 grams	5-15/100 livebirths
Birth Defects	~2/100 livebirths
Chromosomal Abnormalities	
in livebirths	.2/100 livebirths
among spontaneous abortions	35/100 sponteneous abortions
Infant Morbidity and Mortality	
childhood cancer	
severe mental retardation	.4/100 children under 15 years old

* adapted from Warburton, 1981

TABLE 3*

SELECTED CHEMICALS KNOWN TO BE NEUROTOXIC

CHEMICALS	SIGNS AND SYMPTOMS	SITE OF INJURY
Acrylamide	Acute: encephalopathy	Unknown
	Chronic: peripheral neuropathy	Axon
Aliphatic hydrocarbons metabolized to ·diketones	Acute: narcosis	Unknown
	Chronic: peripheral neuropathy	Axon
Arsenic	Chronic: peripheral neuropathy	Axon
Barium	Paralysis	Muscle
Cyanide	Acute: death	Neuron
	Chronic: peripheral neuropathy	Axon(?)
Chlordecone	Tremor, opsoclonus	Unknown
2,4-dichlorophenoxy acetic acid (2,4D)	Peripheral neuropathy	Unknown
Lead	Acute: encephalopathy	Blood vessels
	Chronic: peripheral neuropathy	Axons
Manganese	Acute: encephalopathy	Unknown
	Chronic: Parkinsonism	Basal Ganglia Neurons
Mercury	Inorganic: tremor	Unknown
	Organic: ataxia	Cerebella neurons
	peripheral neuropathy	Axon
Methanol	Acute: encephalopathy	Unknown
	blindness	Retina
	Chronic: blindness	Retina
Organophosphorous compounds	Acute: cholinergic poisoning	Acetylcholinesterase
	Chronic: ataxia, paralysis	Long tracts of spinal cord
	peripheral neuropathy	Axon
Organotin compounds	Memory defects, disorientation, seizures	Neurons of limbic system
	encelphalopathy	Myelin
Polybrominated biphenyls	Memory loss	Unknown
Tellurium Compounds	Prenatal: hydrocephalus	Unknown
	Postnatal: peripheral neuropathy	Schwann cell
Thallium	Acute: encephalopathy	Unknown
	Chronic: cranial and peripheral neuropathy	Axon
Toluene	Acute: narcosis	Unknown
	Chronic: ataxia	Cerebellum
Trichloroethylene	Cranial neuropathy	Axon

* adapted from Grisham, 1986

TABLE 4

EVIDENCE OF CARCINOGENICITY IN HUMANS AND ANIMALS FOR CHEMICALS AND INDUSTRIAL PROCESSES FROM IARC MONOGRAPHS*

Chemical or process	Degree of evidence In humans	In Experimental animals
1. Acrylonitrile	Limited	Sufficient
2. Aflatoxins	Limited	Sufficient
3. 4-Aminobiphenyl	Sufficient	Sufficient
4. Amitrole (aminotriazole)	Inadequate	Sufficient
5. Arsenic and certain arsenic compounds	Sufficient	Inadequate
6. Asbestos	Sufficient	Sufficient
7. Auramine	Limited	Limited
8. Manufacture of auramine	Sufficient	Not applicable
9. Benzene	Sufficient	Inadequate
10. Benzidine	Sufficient	Sufficient
11. Beryllium and certain beryllium compounds	Limited	Sufficient
12. N,N-Bis(2-chloroethyl)2-naphthylamine(chlornaphazine)	Sufficient	Limited
13. BCME and technical grade CMME	Sufficient	Sufficient
14. Cadmium and certain cadmium compounds	Limited	Sufficient
15. Carbon tetrachloride	Inadequate	Sufficient
16. Chlorambucil	Limited	Sufficient
17. Chloramphenicol	Inadequate	No data
18. Chlordane and heptachlor	Inadequate	Limited
19. Chloroprene	Inadequate	Inadequate
20. Chromium and certain chromium compounds	Sufficient	Sufficient
21. Cyclophosphamide	Limited	Sufficient
22. DDT	Inadequate	Limited
23. Dieldrin	Inadequate	Limited
24. Diethylstilbestrol	Sufficient	Sufficient
25. Dimethylcarbamoyl chloride	Inadequate	Sufficient
26. Dimethyl sulfate	Inadequate	Sufficient
27. Epichlorohydrin	Inadequate	Limited
28. Ethylene oxide	Limited	Inadequate
29. Hematite	Inadequate	Negative
30. Underground Hematite mining	Sufficient	Not apolicable
31. HCH (technical HCH and lindane)	Inadequate	Limited
32. Iron dextran	Inadequate	Sufficient
33. Isoniazid	Inadequate	Limited
34. Isopropyl oils	Inadequate	Inadequate
35. Manufacture of isopropyl alcohol(strong acid process)	Sufficient	Not applicable
36. Lead and certain lead compounds	Inadequate	Sufficient(some soluble salts)
37. Melphalan	Sufficient	Sufficient
38. Mustard gas	Sufficient	Limited
39. 2-Naphthylamine	Sufficient	Sufficient
40. Nickel and certain nickel compounds	Limited	Sufficient
41. Nickel refining	Sufficient	Not applicable
42. Oxymethalone	Limited	No data
43. Phenacetin	Limited	Limited
44. Phenobarbitone	Limited	Limited
45. N-Phenyl-2-naphthylamine	Inadequate	Inadequate
46. Phenytoin	Limited	Limited
47. Polychlorinated biphenyls	Inadequate	Sufficient
48. Reserpine	Inadequate	Inadequate
49. Soots, tars, mineral oils	Sufficient	Sufficient
50. Styrene	Inadequate	Limited
51. Trichloroethylene	Inadequate	Limited
52. Tris(aziridinyl)-p-benzo-quinone(triaziquone)	Inadequate	Limited
53. Tris(1-aziridinyl)phosphine sulfide(thiotepa)	Limited	Sufficient
54. Vinyl chloride	Sufficient	Sufficient

Note: Sufficient = "sufficient evidence to support a causal relationship between the exposure and cancer"

* Reproduced from Travis and Cook (1989)

CHAPTER 3

APPROACH TO ASSESSING HEALTH EFFECTS FROM HAZARDOUS WASTE MANAGEMENT FACILITIES

The difficulties in conducting studies to assess unambiguously the health effects associated with population exposure to hazardous waste disposal sites cannot be sufficiently emphasized. These difficulties are discussed at length in numerous publications (eg. Stein et al., 1981; Phillips and Silbergeld, 1985; Levine and Chitwood, 1985; Grisham, 1986; and Marsh and Caplan, 1987a, 1987b). Even with a known chemical inventory and potential population at risk defined, the ability of a study to detect an effect when it is present,(i.e. the "power" of the study) is severely limited by numerous factors, including small population sizes, low background rates of specific diseases, long latency periods, and the non-specificity of many of the potential health effects associated with chemical exposures. Moreover, the actual exposures experienced by a study population may be too low to result in detectable increase in risk, even for known specific health outcomes assessed. As such, as noted above, and further discussed in Chapter 4, the evidence for any health effects associated with chemical waste disposal sites, even those that were extremely poorly contained, is weak at best.

Nonetheless, the science of risk assessments can be brought to bear to address the question of "what is the probability of harm?", attempting to synthesize all available data and the best scientific judgment to begin to provide a perspective to a community considering the establishment of a hazardous waste management facility. As described in detail by Travis and Cook (1989), methodology can be employed to determine health risks from hazardous waste management facilities such that the probability of harm can be expressed in a range between zero and one: zero risk indicates certainty that harm will not occur, while a risk value of one declares certainty that harm will occur. The authors apply these risk assessment procedures to hazardous waste incineration; interested readers are encouraged to examine this publication in great detail for further insight to this particular aspect of hazardous waste management.

This chapter outlines the basic preliminary steps in risk assessment, namely 1)hazard identification, 2) exposure characterization and 3) dose-response or hazard assessment. Actual "risk assessment" for a specific community contemplating specific activities is based on combining this hazard assessment with actual data on the waste stream and likelihood of exposure by various routes given the hydrogeology, population factors and other characteristics.

This process is essential to assessment, but beyond the scope of this "Health Considerations" report.

3.1 HAZARD IDENTIFICATION: HOW WE KNOW A SUBSTANCE IS TOXIC

The first step in estimating the probability of harm, given a worst case scenario, is to collect and evaluate information concerning the inherent toxic properties (potential to cause biological injury, disease or death under certain exposure conditions) of chemicals to which potential exposure may occur. There are basically four sources of data which can be used: human studies, (including both formal epidemiological studies as well as information from accidental human exposures), animal tests, short-term laboratory tests, and comparison of molecular structure. Each of these will be discussed briefly below:

3.1.1 Human Studies: Epidemiology, defined as the study of the distribution and determinants of disease, can be divided into two main types: descriptive and analytical studies. Descriptive studies include case reports, case series, as well as reports of incidence (new cases) or prevalence surveys of disease in particular populations. These surveys can be conducted as outbreak investigations, surveys requested by the community, or planned surveillance. Reports from individual cases or series of cases are based on clinical encounters, with no defined population base, and therefore provide only speculative evidence of causation. Surveys of communities that have had particular exposures, may include investigations for acute clinical symptoms, laboratory abnormalities or more serious disease. While able to detect the prevalence of current concerns or the incidence of events (e.g. miscarriages), these surveys are often based on outcome which can be considered "subjective" or of questionable clinical validity. Moreover, descriptive studies are just that, descriptive. As there is no comparison population or baseline with which to compare the outcome, the link to the exposure is still considered speculative.

Even when there is a control group, surveys or "cross-sectional studies" are subject to "recall bias" or "reporting bias". In other words, respondents in the target area may give special importance to certain symptoms. For example, a respondent who believes his headache resulted from breathing chemicals may remember his headache better or be more prone to mention it than someone for whom a headache is just a minor annoyance of no particular consequence. Unfortunately, recall and reporting bias tends to increase with proximity to the source of exposure, just as one would expect biological effect to increase. Of course, people who believe their headaches result from breathing chemicals may very well be correct. There are ways to test for recall or reporting bias, for example by assessing the effect of the exposure variable only on

individuals who answer "no" to a question of whether they believe the air or water in the neighborhoods made them ill. However, this method has its obvious drawbacks.

Analytical epidemiological studies, on the other hand, are designed to provide better evidence on causation. The two main types of analytical studies consist of the cohort and case-control study. Cohort studies assess whether particular health outcome (specific diseases or increased mortality from a specific cause) are different in two populations with different exposures. Case-control studies contrast people having a particular disease with people who do not have that disease, in order to assess whether more people who have the disease had the exposure in question compared to those who do not have the disease. The major strengths and limitations in the various types of epidemiological studies are discussed at length in the publications cited at the beginning of this Chapter. In general, given identical samples sizes, the cohort design is more powerful when the exposure is less common than the outcome, and the case-control is preferable if the outcome is rare. In both designs, however, it is important to look for confounding variables, in other words, other exposures or characteristics that are also different between the study and the comparison groups which may bias the results one way or the other. Selection bias, i.e. how the study and comparison groups are defined, measurement biases, i.e. how exposure is assessed, and ascertainment biases, i.e. how the outcome is defined and ascertained are also crucial factors, as discussed at length in epidemiological textbooks.

The success of epidemiological study necessitates valid and accurate information regarding both the chemical exposures, ("independent variables"), as well as the biological abnormality or disease (the "dependent or outcome variables") . Both of these are usually difficult to achieve in environmental health studies, especially in studies of chemical waste disposal sites. Moreover, even with relatively good data on exposure and target outcome of concern, often the disease in question, eg. cancer, has a large latency period, i.e. takes many years to develop. While there is some variability, generally if the exposure occurred less than 10 to 20 years previously, it would be unusual to find an increase in cancer. It must also be borne in mind that the outcomes are almost always nonspecific, in other words, could have been caused by a multitude of factors either separately or in combination. Four further parameters must be considered, as they influence statistical power (the likelihood of finding a true difference at a level that would not be considered a chance happening): the larger the number of persons studied, the greater the power; the unknown variable must be sufficiently common in the unexposed or unaffected group; the greater the size of the association between exposure and outcome the greater the chance of detecting the increase; and, finally, the improbability of a

chance finding (or "p" value) that will be considered by the investigators as "significant" will determine the power of the study to detect a "significant" result.

Before a link between exposure and effect are accepted by the scientific community as truly "causal" a set of criteria have to be met. These include a) adequate study design, b) consistency of association across several studies, c) strength of association, i.e: marked differences between exposed and unexposed, d) temporality, i.e: evidence that the disease or effect did not precede the exposure, e) specifity of the outcome, f) coherence, or biological plausibility of the findings, g) dose-response relationship, such that the effect is greater among those more exposed, and h) that the finding makes epidemiological sense with what is already known.

Because properly designed epidemiological studies can be expensive and time consuming, much of the evidence on the toxicity of chemical exposures does not come from studies in humans at all. Table 4 listed the chemicals and processes for which epidemiological and animal data show evidence of carcinogenicity in humans. Note that only 13 chemicals and 5 industrial processes have been identified as having sufficient epidemiological evidence to be classified as definite human carcinogens. In considering all available data, the International Agency for Research on Cancer (IARC) concluded that at most 54 chemicals and industrial processes could be considered as potentially carcinogenic to humans (IARC, 1979). Since that time other agencies and associations have expanded the list somewhat, but as of 1985, the U.S. Environmental Protection Agency (EPA) has provided cancer potency assessments for only 62 chemicals. The evidence regarding specific chemicals likely to be of concern to a community considering establishing a hazardous waste management facility are discussed further in Chapter 5.

3.1.2 Animal Studies: As much of the evidence on toxicity, particularly carcinogenicity, comes from animal study, it is important to recognize some of the limitations of this data. The first problem is that there are indeed species-to-species differences. Some substances known to be carcinogenic in rats do not appear to be carcinogenic in mice, hence inferring carcinogenicity to mammals or particularly to humans is suspect. Secondly, many issues arise in extrapolating from high dose exposure in animals to low dose exposure in humans. As the lifetime of animals is much shorter than that of humans, and as the numbers of people potentially exposed would be far larger than the number of animals that could be studied in laboratories, it is necessary that much higher doses be used in animal studies than humans would likely encounter in the environment, even in a worst case scenario.

3.1.3 Short-term Tests: Short-term laboratory tests have been very useful in evaluating toxicity, and particularly carcinogenic potential. These tests include those in which the suspected carcinogenic substance is applied to a culture medium in a laboratory plate to see if it causes a mutation which can be seen by changes in bacterial growth. These short-term tests can investigate not only gene mutation but also chromosome damage, and these tests could be further enhanced with actual tissue substances.

3.1.4 Comparison of Molecular Structure: Because of the difficulties in interpretation of animal tests and short-term test results, a presumption exists that a chemical which is structurally similar to a known carcinogen has a high probability of itself being a carcinogen. Although this is a prudent approach, the evidence supporting such associations is still quite limited.

As the evidence from epidemiological studies, animal experiments, short-term laboratory tests and molecular comparisons all have serious limitations, establishing proof of hazard, let alone providing information for a dose-response estimate is extremely difficult.

3.2 EXPOSURE ASSESSMENT: HOW WE ESTIMATE WHO IS EXPOSED AND TO HOW MUCH

The second component in risk assessment is exposure analysis. As noted elsewhere in this report, there is no risk without exposure. The exposure assessment component of risk assessment attempts to define the route, magnitude, frequency, and duration of exposure of a given population, and considers exposure through water contamination, ingestion of contaminated food, airborne exposures, and direct contact. Many authorities believe that the weakest link in our understanding of environmental health studies is our knowledge of human exposure (Travis and Cook, 1989). Although measurement of biological indicators in humans would provide the most direct estimate of exposure, this is rarely available. Even environmental monitoring results are often available only after the fact, usually once the problem has largely abated. Another method which has been developed in light of the situation is mathematical modeling. These two methods are elaborated upon briefly.

3.2.1 Environmental Monitoring and Exposure Registries: Landrigan (1983) describes the need for registries of exposed individuals, preferably with individual exposure measurements recorded. Without knowledge of what constitutes the exposed population, little headway will be made in evaluating all the facts available for such exposure. Even if personal monitors were to be used, it should be noted that the majority of such monitors give data for average exposures,

but do not provide information recording exposure times and peak levels. In a study of two New Jersey cities, individuals were continuously monitored for exposure to twenty atmospheric organic chemicals (Wallace et al., 1985). Due to changes in activity patterns throughout the day and night, monitored exposure levels fluctuated greatly, with indoor exposures greatly exceeding outdoor exposures. Thus stationary monitoring of only outdoor pollutant concentrations would have underestimated total exposure. Not only is there variability in health outcome with variable concentrations, there would be variability in exposure from other routes, depending on variation in diet and in a large number of other factors.

3.2.2 Modeling: Mathematical models can estimate human exposure levels where monitoring data are either unavailable or incomplete. Because these models are crude, they are often designed to illustrate worst case scenarios, to err on the side of caution. The basic processes that control the environmental fate of pollutants, namely transport, transformation and inter-media transfer, are all taken into account. Transport is the movement of pollutants within the environmental media of air, water and soil, due to natural forces. The direction and speed of winds, for example, determines where the greatest concentration of an airborne pollutant would be found; for surface and groundwater, transport mechanisms involve study of the movements of sediment carrying the chemicals. Transformation is the process which causes change to take place in the physical and chemical nature of the pollutant. This may involve the interaction with other pollutants, for example, the production of ozone from the effect of sunlight on nitrogen oxides and hydrocarbons. Intermediate transfer is the movement between the environmental media of air, water, soil and plants. For example, an airborne pollutant can be transferred to the earth surface through gravitational settling or wash out by rain. A pollutant in a river may transfer to the ambient atmosphere through volatilization; a pollutant in soil may volatize into the atmosphere or leach into groundwater. Cross media transfer can result in greater potential for human exposure. Uncertainties exist in exposure models because of imprecision in the descriptions of their physical and chemical processes as well as uncertainties in the parameters used as input to the models. Thus there is still considerable controversy in the use of this technique (Travis and Cook, 1989).

3.3 DOSE RESPONSE EVALUATION AND RISK ASSESSMENT: HOW WE DETERMINE PROBABILITY OF HARM

Once the toxic substances and their possible health effects are determined, and once the extent of exposure has been characterized, the next step is to estimate possible human response to that exposure. For some toxic substances, a threshold is assumed to exist, in other words a certain

level of exposure must be exceeded before health effects will occur. For other substances, including all potential carcinogens, the prudent approach, which is generally accepted, is to assume that no threshold exists. Nonetheless, the magnitude of the risk at low levels of exposure has long been highly contentious (eg. Maugh, 1978).

The US EPA has calculated cancer potency factors for 62 hazardous constituents, using the most conservative or cautious estimates to indicate the probability that an individual will develop cancer after a 70 year exposure to the chemical at a dosage of 1 mg/kg body weight per day. These numbers are shown in Table A of Appendix 6.

The next step in the risk assessment procedure is the determination of exposures and doses. As discussed above, this is a function not only of the levels of emissions, but also air dispersion, exposure duration, body weight, absorption, inhalation, ingestion and other factors, discussed at length by Travis and Cook (1989) and others. To determine the population risk, this is multiplied by the number of people residing in that exposure sector, added to the numbers from sectors of lesser exposure and so on to quantify the population exposure.

For non-carcinogenic constituents of hazardous waste streams, the "allowable daily intake" (ADI) is used as a measure of chronic toxicity. This is also discussed in Appendix 6. An ADI is defined by the EPA as the amount of a toxic agent in mg/kg of body weight per day which is not expected to result in any adverse effects after chronic exposure in the general population of humans, including sensitive subgroups. Because of uncertainty, safety factors are used. A safety factor of 10 is used when the "no observed adverse [chronic] effect level" (NOAEL) is based on human data, to account for variability within populations; a safety factor of 100 is used when the NOAEL is based on data for chronic effects in animals, to account for both intra- and inter-species variability; a safety factor of 1000 is used when the NOAEL is based on sub-chronic animal data, to account for uncertainties in extrapolation, in addition to the previous uncertainties noted. Table B of Appendix 6 presents EPA estimates of ADI for the chemical constituents of waste currently incinerated in some parts of the U.S. If the estimated daily intake is less than the ADI, there should be no adverse human health effect from continuous exposure to the chemical.

Aside from the uncertainties related to high dose to low dose extrapolation, interspecies extrapolation,and mathematical models, hazardous assessment does not allow for such factors as different dose response levels among children, adults and the elderly, as well as differences in

physiological factors within populations. It also fails to take into account that two or more exposures can interact to create a disproportionately greater effect.

The final steps in risk assessment, the process to answer the question of "how much harm is probable" is to combine the above information with specific information on possible exposures to be encountered in the actual hazardous waste management facility in question. This requires integrating information from this report with that obtained from projections of the waste streams and other relevant data.

CHAPTER 4

HEALTH FINDINGS IN STUDIES OF TOXIC WASTE DISPOSAL SITES

The management of industrial wastes is not a new problem and Tarr has described how its serious nature was recognized in the United States more than a hundred years ago (Tarr, 1985). While the problem has long been recognized, the demonstration of its relationship to adverse health effects has been difficult. The gross anatomy of the condition has been described and statistics are available which show that in 21 counties of New Jersey, mortality from cancers of the gastrointestinal tract is higher than in any other part of the United States (Najem et al., 1983). The environmental variables most often associated with gastrointestinal cancer mortality were urbanization, population density and chemical toxic waste disposal sites. New Jersey is a centre of the chemical industry and has been heavily industrialized for more than a century. Until recent years, attempts at pollution control have been limited and the management of industrial wastes largely unregulated.

There are two aspects of the toxic industrial waste question. The emissions from an industrial process arise from a point source and may be rapidly broadcast by air and water throughout the environment. The wastes of a process, those which do not form part of an effluent, but for which there is no further practical or economic use by the organization that creates them, are gathered together and disposed of in a manner that is either controlled or uncontrolled. Depending upon the degree of management imposed upon these wastes and upon their toxicity, they too may add to the pollution hazard confronting the general population. It is with the latter type of waste and its health effects rather than with emissions, that this discussion will be concerned.

In a heavily populated and industrialized area, the community will be exposed to hazards arising from both types of waste and there is difficulty in determining to which a particular adverse health effect may be attributed. With few exceptions such as the causal role of asbestos in the induction of mesothelioma of the pleura and peritoneum (linings of the lungs and abdomen respectively), cause-effect relationships are non-specific and it is not possible to say

with certainty that exposure to a given agent has caused the occurrence of a specific disease such as leukemia or cancer of the gut.

There are now in excess of 160 licensed hazardous waste management facilities in the U.S. as well as approximately 1,000 designated as Superfund priority sites. There also also numerous European facilities and at least four licensed hazardous waste management facilities in Canada. These sites all provide candidate areas for public health concern. Those which have indeed become sources of significant public health concern are discussed in this report.

4.1 EPIDEMIOLOGICAL LIMITATIONS RE-EMPHASIZED

As discussed in Section 2.2, diseases known to be associated with occupational and environmental exposures indicate the sort of adverse health effects that may appear in populations living near hazardous waste management sites. As was discussed in Section 3.1.1, epidemiological studies of people in such areas are made difficult by the low statistical power associated with neighborhood-sized populations, by poor measures of exposure, the lack of well-defined endpoints and by the migratory instincts of populations, both human and animal. These problems are compounded by the difficulties inherent in assessing the significance of disease clusters that may occur in the vicinity of specific sources of pollution. The occurrence of clusters of childhood leukemias in the neighborhood of some nuclear installations has been described (Black 1987), but definite linkage of any of these sources with a specific agent has not been possible and factors such as infection that may confuse the issue have been proposed (Delamothe 1989). Attention has been given to the problem of clustering in two recent publications (COMARE, 1989; Leech, 1989) and it is important to view reports of disease clusters in the perspective of the recommendations made.

The acute effects of exposure to materials contained in hazardous waste management sites may be more easily identified than long-term effects such as cancer and genetic detriment, but respiratory symptoms, skin lesions, eye troubles, and gastrointestinal disturbance, effects that are commonly reported in relation to these exposures occur frequently in the general population and are usually non-specific insofar as causation is concerned. Most epidemiological studies of waste management sites are started long after the time at which acute effects might be expected. Indeed the coverage of known sites is sparse and by 1985 in the United States, fewer than 20 of some 900 EPA listed sites had been the subject of clinical epidemiological investigations. In most

of these studies, only severe health outcomes such as cancer and serious birth defects have been investigated (Phillips and Silbergeld, 1985).

Reference has been made earlier to the fact that diseases which have a recognized association with occupational exposure give useful leads to the identification of adverse health effects in a community exposed to similar toxic agents. This being so, studies of populations occupationally exposed to the hazards of toxic wastes would be of great interest even if of limited statistical power. Unfortunately, few such reports are available. Dewolfe-Miller and colleagues have described their study of Zabaline (solid waste scavengers) in Egypt (Dewolfe-Miller et al., 1982). Their findings of parasite infestation, trauma and elevated infant mortality rates are of undoubted importance to these poor people, but cannot be applied to the generality of those who work in waste management operations. A small study of workers exposed to pulverized fuel ash is possibly applicable to the occupational health aspects of hazardous waste management (Schilling et al., 1988). Effects on lung function were identified that confirmed the need for control of exposure to such ash. Scarisbrick (Scarisbrick, 1986) has described the occupational health and safety (OHS) aspects of a reclamation project, the problems of which had much in common with those of waste management and lend emphasis to the need for good OHS programs to be operated at waste management sites, as described in Section 2.1.

As noted earlier, it is generally held that studies of occupationally exposed populations are significant in the sense that if no adverse effects are found in such groups, it is improbable that adverse effects will exist in the context of community exposure. As Hughes and Schwartz have suggested, however, "the inability to measure adverse health effects does not mean that they do not exist. Rather, it means that the state of scientific knowledge is such that we cannot demonstrate their presence with reasonable certainty" (Hughes and Schwartz, 1983). Nonetheless, studies of the working populations at engineered waste management facilities in West Germany (Ebenhausen) and Denmark (Nybong) have not led to the identification of any abnormality in these populations (Weeks, 1989 personal communication).

In determining the nature and extent of possible adverse health effects that may be related to the management or mismanagement of toxic wastes, the epidemiological data now available do not permit the achievement of firm conclusions and it is improbable that this picture will change greatly during the next decade. Two conclusions can however be reached, namely, that toxic substances can only harm those who are exposed to them and one does not

need a sophisticated epidemiological study to know that exposure to chemical toxins is unlikely to be beneficial.

Adjuncts to epidemiological studies such as air monitoring, the monitoring of food, water and breast milk in the vicinity of waste management sites have a role to play in leading to the identification of adverse health effects. The monitoring of animals, not discussed extensively in this report, also has a role to play. The measurements of birthweights and growth during infancy and childhood have provided some of the few firm indicators of human effect for chemical waste. Cytogenetic studies and, in particular, the identification of sister chromatid exchanges (SCE) offer a more sensitive way of assessing damage in a potentially exposed population, as was discussed in Section 2.2. As pointed out by Wolff (Wolff 1983), even when cytogenetic tests on peripheral lymphocytes show that genetic material has in fact been damaged, these observations can only be used to estimate risk in a population, they cannot be used to predict that any individual will suffer a particular form of ill health. Similarly, mutagens are not necessarily carcinogens and mutagenic tests are designed to indicate genotoxic potential; they will not identify foreign body carcinogens, carcinogenic hormones, immune suppressors or co-carcinogens (Tassignon 1985).

4.2 ADVERSE HEALTH EFFECTS AND UNMANAGED WASTE DISPOSAL SITES

As emphasized throughout this report, an examination of the adverse health effects that may occur in the vicinity of a waste management site is attended by many uncertainties. Hazardous wastes can be subjected to varying degrees of control which range from merely tipping them into a hole or discharging them into the nearest waterway, to their processing and containment in adequately engineered waste management facilities. There are many intermediate steps in this spectrum, but until recently, inadequate or absent engineered control has been characteristic of most waste management practices and this circumstance has led to many occasions on which adverse health effects have been reported in exposed populations. Few such effects have been confirmed by epidemiological studies.

The Love Canal is probably the most well known waste dump in the world and there is an extensive literature dealing with health problems in the vicinity of this site, some of which are outlined in Appendix 2. For example, one carefully controlled study of cytogenetic abnormalities noted no differences between exposed and control subjects in the frequency of

genetic abnormalities, including chromosomal aberrations and sister chromatid exchanges, (Heath et al., 1984). An ecological study which examined incident rates of cancer in the Love Canal area showed no excess compared to the rest of the State of New York for any cancer, except possibly lung cancer, which was inconsistent (Janerich et al., 1981). In review of other clinical investigations, it was concluded that there were no unusual patterns noted for a wide range of conditions, except possibly for an increased frequency of low birth weight in babies born to women from homes with possible seepage risk, however the finding did not correspond with the environmental test results (Heath, 1983).

In reviewing the reports available prior to 1985 Grisham (1986) has written: "Although these and several similar studies do not confirm a causal association, they cannot be interpreted to indicate that no risk exists, but rather that no serious health effects clearly attributable to a specific chemical or group of chemicals have been identified to date by the study methods employed." Implicit in this statement is recognition of the fact that the latent period for most chemical carcinogens has by no means expired in the Love Canal exposed population, nor is there any way by which the eventual cancer experience of that population can be estimated with reasonable accuracy.

Since preparation of Grisham's book (Grisham, 1986) some further information has become available about the Love Canal residents. Cuddy and her colleagues have reported on a small pilot study in which theophylline plasma clearances were compared in 11 Love Canal residents and 25 control subjects from Western New York State, and as noted in Appendix 2, found to be similar (Cuddy et al., 1984). Theophylline clearance was used in this study as an indicator for the integrity or otherwise of one pathway of microsomal P-448 oxidation, thereby constituting a sensitive and sophisticated liver function test. The authors define carefully the limitations of their study, including the fact that none of the subjects were among the more heavily exposed of the Love Canal residents, who had already been evacuated from the area when the study was done. Vianna and Polan (1984) also studied birth weights in the Love Canal area during the period 1940-1978 and found that there was a significant excess of low birth weights in the area of an historic swale that formed part of the natural drainage system of the chemical disposal site. The excess was limited to the period 1940-52 during which disposals were being made in the area. In a subsequently reported study, a high prevalence of low birth weights was confirmed in a wider section of the Love Canal population and it was proposed that low birth weight is a sensitive indicator of adverse effect that could be used in other populations

where exposure to toxic wastes was suspected (Goldman et al., 1985). In this study, birth defects were also found to be increased in one of the two subdivisions of the exposed population. Along similar lines, it was found in a later study that children born and spending at least 75% of their lives in the Love Canal area had significantly shorter stature for age than control children (Paigen et al., 1987).

The incidence of lung cancer in nine census tracts (including the Love Canal area) containing 12 toxic waste sites was again studied (Polednak and Janerich, 1989). No significant association was identified between lung cancer and a history of ever having resided in the selected census tracts. The limitations of this study were discussed by the authors who recommend that the negative findings should be interpreted with caution.

While the Love Canal is a well known example of toxic waste mismanagement, others have been identified. A toxic waste dump at Hardeman County in Tennessee was closed in 1972 when it contained some 10 million liters of solid and liquid wastes. While there were no records of what was put into this dump, leakage from the dump into surface and underground aquifers was thought to have led to the contamination of drinking water with chlorinated pesticides which was found. Evidence of impaired liver function was subsequently gathered from the exposed population (Meyer, 1983). Although no chlorinated organic chemicals or metabolites were detected in the urine of any of the residents, liver function tests (specifically alkaline phosphatase and SGOT) were elevated in exposed individuals. Medical examination showed no significant differences between high, low and medium exposure groups, except for borderline hepatomegaly (enlarged liver). A subset of the residents were retested three months later and it was found that their liver function test had returned to normal levels. The authors concluded that mild liver injury probably did occur in the population as a result of drinking the contaminated water but that this injury was transient and had resolved (Clark et al., 1982).

When an event can be investigated shortly after its occurrence, a different approach to the demonstration of exposure can be taken. In 1979, 1900 gallons of trichloroethylene (TCE) were released into the ground and surface water in Montgomery County, Pennsylvania. It was subsequently possible to identify TCE metabolites in the urine of community residents and of those who had been occupationally exposed (Landrigan et al., 1987).

The Drake Superfund Site in Clinton County Pennsylvania, was used for the storage of large quantities of waste organic chemicals, including -naphthylamine and benzidine. A significant increase of bladder cancer deaths occurred among white males in Clinton County, together with a significant increase of other cancer deaths in the general population of Clinton and three surrounding counties (Budnick et al., 1984). It is possible that male occupational exposure was in part the cause of the increased incidence of bladder cancer. The New Jersey experience has been cited in the introduction to this chapter and is further developed by the identification of increased cancer mortality rates of female reproductive organs and breast in that state (Najem and Greer, 1985). These studies do not relate to point sources of contamination, but to a more general pollution of the environment.

A particularly interesting, but unsigned paper appeared in the American Journal of Law and Medicine (Anon, 1987). In this paper is described a cluster of six cases of childhood leukemia occurring at Woburn, Massachutts. Although toxic waste problems at the town were obvious, it was not possible to demonstrate a causal link between exposure to toxic wastes and the incidence of leukemia at Woburn. This difficulty, inherent in the problems of cluster epidemiology, plagued and continued to plague studies of toxic wastes and health effects. Although the initial investigators felt that the evidence establishing an association between the environmental hazards and the increased incidence of childhood leukemia and renal cancer was insufficient, other investigators, conducting a separate study, concluded in an unpublished undated report that a consistent pattern of childhood leukemia, perinatal deaths and certain birth defects and childhood disorders was indeed associated with the availability of water from the contaminated wells (Buffler et al., 1985). The Woburn findings were recently followed-up in exposed subjects and alterations in the immunological system, specifically in the T-lymphocyte subpopulations, were found (Byers et al., 1988).

It is possible that endpoints other than cancer may be useful in identifying the impacts of exposure. As discussed in Section 2.2, an increase in the frequency of many common medical complaints has been reported from Lowell, Massachusetts, where extensive and repeated spills of organic wastes had occurred on the property of a company since bankrupted (Ozonoff et al., 1987). As was explained in Section 2.2, the authors of this study accepted that the self reporting of a symptom is in itself an 'objective fact', a viewpoint which is important in this approach to the determination of endpoints.

When the leachate from the abandoned chemical waste disposal site in Chester Pennsylvania caught fire in February 1978, firefighters reported cough, headache and skin rash as major symptoms resulting from contact with smoke and fumes at the site. This episode lead to the investigation of 31 households, including 86 residences around the dump to detect possible illnesses associated with the site. Although most were aware of the waste site, none reported any ill effects associated with it. Fourteen neighborhood children were examined, and showed no unusual dermatological, nervous or liver function abnormalities (Buffler et al., 1985). The symptoms reported by the firefighters were consistent with those reported in fires generally, and may not have been due to specific toxic exposure.

Appendix 2 outlines other investigations of unmanaged or mismanaged chemical waste disposal sites.

From the foregoing it is apparent that Grisham's statement to which reference has earlier been made requires little revision. The evidence from Love Canal insofar as it relates to low birth weights, reduced stature, and birth defects can not be ignored. The elevated incidence of leukemia at Woburn is supported by a later finding of altered subpopulations of T-lymphocytes in the exposed population. Although this cannot be considered proof of a causal link, it is certainly noteworthy. The known role of -naphthylamine and benzidine in the causation of occupational bladder cancer was once again confirmed in Clinton County where occupational as well as environmental exposures were common. These and similar observation point in the direction of a link between adverse health effects and exposure to contamination from ill-managed toxic waste disposal areas.

4.3 ADVERSE HEALTH EFFECTS IN THE VICINITIES OF "MANAGED" TOXIC WASTE DISPOSAL SITES

A managed toxic waste disposal site may be simply defined as one in which adequate engineering procedures have been applied to ensure that the wastes contained in the site remain contained and are not permitted to contaminate the environment. There are varying degrees of management and the waste management site at Ebenhausen/Gallenbach in the Federal Republic

of Germany is a good example of the intelligent application of engineering technology to the management of toxic wastes (Defregger 1983).

Few reports concerning adverse health effects around managed waste disposal sites are available. This may be because the job has been well done and there are no effects to detect, or possibly the fact that engineered waste management is a comparatively recent phenomenon means that long-term adverse effects have not yet had time to become apparent. Many of the effects that have been reported refer to the incineration of wastes. The effects of the incineration of household and industrial wastes at Isere (France) have been indirectly measured by the purchase of medication for respiratory problems (Zmirou et al., 1984). This measure is objective in the same sense as that described by Ozonoff (1987) and there was a significant decrease in the number of medicines prescribed as the distance of the residents' home from the incinerator increased. It was not possible to describe the precise nature of effluent from this incinerator, but following the presentation of the report, measures were taken to reduce emissions.

An increased rate of twinning in human populations around two incinerators in central Scotland has been reported (Lloyd et al., 1988), but this finding was subsequently refuted (Jones 1989) and the issue is unresolved.

A study of cause of death among workers at a municipal waste incinerator near Stockholm (Sweden) has provided evidence of increased mortality due to ischaemic heart disease and possibly lung cancer, but no community effects were reported (Gustavesson 1989).

A recently reported investigation into health effects in relation to the location of waste incinerators in the United Kingdom has lead the authors to conclude that the available data are inadequate to determine any significant correlation (Gatrell and Lovett, 1989). A recommendation was made that better mortality and environmental data should be developed in the vicinity of incinerators. Karasek and Hutzinger have drawn attention to the technical aspects underlying dioxin emissions from incinerators, but this paper did not extend to observed health effects that might be attributable to dioxin exposure (Karasek and Hutzinger, 1986). Similarly, as described by Mohr (1988), an international dioxin symposium held in Bayreuth West Germany concluded that all municipal solid-waste incinerators produce hundreds of organic compounds including dioxins and furans and that emissions may travel huge distances. Again no health effects were documented. In one incident, Danish pigs fed downwind from a garbage

incinerator produced a litter with a high rate of deformities. A cause-and-effect relationship was never established, but public outcry resulted in the closing of the facility.

The Stringfellow Hazardous Waste Disposal Site is a Class 1 industrial waste disposal facility located in California. During the period of its use, the containment ponds had overflowed on several occasions and the site was closed in 1972. Overflows occurred after closure and sampling of surface waters following episodes in 1977 and 1978 showed that wastes had moved up to four miles from the site. The health study of the affected area and comparison with a reference population showed no differences with respect to mortality, cancer incidence and pregnancy outcome, but there was a marked increase of short-term adverse health effects in the study area (Baker et al., 1988). The observations are in keeping with the temporal considerations applicable to the occurrences at Stringfellow and relate to a site at which the provisions for waste management proved to be inadequate.

Thus the reports of adverse health effects from managed waste disposal sites relates mainly to incinerator emissions with the exception of the Stringfellow report which noted short-term health effects possibly attributable to overflows in the containment pond. As specific health effects are best considered in conjunction with the actual waste stream, the next chapter discusses the toxic effects of specific chemicals.

CHAPTER 5

TOXICITY OF INDIVIDUAL COMPOUNDS

The purpose of this chapter is to describe potential adverse health effects which may occur following exposure to a variety of chemical compounds. The individual compounds discussed are either currently present in Manitoba's waste stream or are of such high public concern to warrant inclusion. Both acute and chronic problems associated with exposures to organic and inorganic compounds will be covered. Although the majority of evidence regarding adverse effects from exposure to chemical compounds is obtained from studies of workforces, usually at levels much higher than those sustained in environmental exposures, when available, environmental exposure data will be presented to discuss the effects hazardous waste sites or accidental exposures could have on neighboring communities.

5.1 - INORGANIC COMPOUNDS

5.1.1 Lead: Lead has been used for thousands of years and is a compound for which a great deal of toxicologic and epidemiologic information is available. Lead enters the body through inhalation and ingestion. It is stored in soft tissues and later bones, and is slowly released from these stores to be excreted by the kidneys. Once lead has been stored in bones its half life, the length of time required to eliminate half of the lead load, may be many years.

Extremely high levels of lead can lead to lead colic (severe cramps) or coma. These effects are only likely to occur after uncontrolled occupational exposure or following ingestion of large amounts of this metal. For people living around contaminated hazardous waste sites only children who ingest soil heavily contaminated with lead would be at risk for serious complications.

Lead effects a number of organ systems including blood forming organs, peripheral and central neurologic systems, the kidneys, and the reproductive system. The degree of effect that lead has on each of these organ systems is dependent on the extent of exposure. Lead can be measured in both the blood and urine, although blood lead

measurements are more useful. Currently the Manitoba standard for removal of workers exposed to lead from working with lead is 50 μg/dl (micrograms per decilitre) of blood. Urban dwellers without occupational lead exposures usually have blood lead levels around 10 μg/dl (Bellinger et al., 1987).

High levels of lead can lead to anemia, or low red blood counts, by inhibiting the production of red blood cells and by increasing the rate of their destruction. Two of the enzymes important in the synthesis of hemoglobin, the constituent of blood that carries oxygen, are inhibited at relatively low levels of blood lead. Although these enzymes are inhibited by low blood lead concentration, and at levels that can affect the rate of blood regeneration, anemia only occurs at higher blood lead measurements (Grandjean et al., 1989). Changes in these enzymes (eg. free erythroyte protopophyrins or FEP) can be used to assess early effects of lead exposure.

Lead's effect on the peripheral nervous system is also well known. High levels can lead to wrist drop, a condition where the wrist cannot be elevated. Lower levels of lead under 40 μg/dl have been found to cause slowing of nerve conduction velocities in asymptomatic individuals (Seppalainen, 1980).

The central nervous system effects of lead include fatigue, loss of memory, irritability, and sleep disturbance. Although individuals with blood lead levels above 60 μg/dl routinely show these symptoms, individuals may be symptomatic at lower levels. Decrements in neuropsychological testing have been found at lower blood lead levels. Still lower levels of lead have been shown to effect neurologic development of children. This is particularly concerning because symptoms suggestive of lead toxicity may not be present (Landrigan, 1989).

High levels of lead have been associated with kidney failure. This finding though has not been reported at lower levels of exposure. Although high dose lead exposure has been associated with high blood pressure, whether lower exposure levels will lead to this condition is an area of current research (Pirkle, 1985).

Lead had also been shown to be toxic to the reproductive system of both men and women in occupational settings. These studies have reported blood lead levels in the

greater than 50 g/dl range (Landrigan, 1989). A very concerning fact regarding the toxicology of lead is that babies born to women with blood lead levels as low as 15-20 g/dl have shown worse neurologic scores on testing than unexposed children (Bellinger et al., 1987)(McMichael, et al., 1988).

Lead toxicity either due to ingestion of contaminated water or soil or living near lead smelters have been extensively reported (eg. Roels et al., 1989; Clarkson et al., 1983). Aside from the situation where children ingest heavily lead contaminated soil, lead exposures around hazardous waste sites are likely be of low dose and chronic. Blood tests are available which can evaluate the extent to which an individual is being over-exposed to lead.

5.1.2 Cadmium: Cadmium is widely used in the electroplating industry to make other metals corrosion resident . Extensive amounts of cadmium are also used in the production of pigments (Friberg, 1983).

Inhalation of extremely high levels of cadmium fumes may lead to chemical pneumonitis or pulmonary edema (Barnhart, et al., 1986). Chronic lower level exposures to cadmium fumes may lead to emphysema.

In individuals not occupationally exposed to cadmium food and tobacco smoke are the major sources of cadmium exposure (Bernard 1984).

Ingestion of rice grown in areas of Japan environmentally contaminated by cadmium has led to Itai-Itai ("ouch-ouch") disease (Tsuchiya 1978), a condition caused by a cadmium induced abnormality in bone metabolism. Elevated mortality from chronic renal failure has been reported in this area (Nakagawa 1987), as noted in Appendix 3.

Cadmium can be measured in the blood and urine. Early markers of cadmium induced kidney disease are available (Clarkson et al., 1983).

5.1.3 Chromium: Chromium is principally used in chrome plating to enhance beauty and increase corrosion resistance. Chromium exists in two valant states, trivalant and hexavalant. The trivalant state is much less toxic than the hexavalant state (Landard and Norseth, 1986). Hexavalant chromium is very irritating and corrosive. Contact with high concentrations of chromium can lead to damage either to the skin or respiratory tract (Bidstrupp 1983). Hexavalant chromium is also known to be a pulmonary carcinogen (Yassi and Nieboer, 1988).

5.1.4 Nickel: Specific nickel compounds are, in fact, now known to be carcinogenic (Nieboer and Yassi et al, 1983), and are currently being regulated as such. It is an essential metal and is found in human proteins and enzymes. Nickel however is also toxic following exposure to elevated concentrations. Nickel carbonyl, produced when nickel is reacted with carbon monoxide, is severely toxic to the lungs and can lead to pulmonary edema. Other nickel compounds may cause damage to the respiratory tract due to their irritant properties. Nickel is also a potent allergen and can cause allergic contact dermatitis and asthma in susceptible individuals (Norseth 1986)).

Nickel refinery workers have been shown to have elevated risks of lung and nasal sinus cancer (Roberts 1980). Nickel is normally present in water and soil, the exact concentrations being dependent on the mineral content of nearby soil.

5.2 - ORGANIC COMPOUNDS

5.2.1 Cyanide salts: Cyanide salts are used extensively in electroplating industries. In their salt form these compounds do not vaporize but can be absorbed through the skin. When mixed with acids cyanide salts are converted to hydrogen cyanide which enters the body through skin absorption or by inhalation. Cyanide compounds inhibit the body's ability to utilize oxygen causing, in extreme situations, death by asphyxiation. Chronic low grade ingestion of cyanide salts can lead to thyroid gland abnormalities and peripheral neuropathies (Hardly 1983).

Cyanide is found in the pits of certain fruits, such as apricots and also is a product of cigarette smoke. Cyanide poisoning is a well known complication of the use of Laetril, an alleged anti-cancer drug.

5.2.2 Dioxin: Dioxin, 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) has received a tremendous amount of attention due to its high level of carcinogenicity in experimental studies. The degree to which this compound causes cancer in humans has been an area of intense scientific research. Some but not all occupational studies have shown that it causes soft tissue growths called sarcomas and non-Hodgkin's lymphomas (Eriksson et al., 1981, Hardell et al., 1981, Hoar et al., 1986). A great deal of information regarding the human toxicity of dioxin has been obtained from studying individuals exposed to this compound following an environmental accident in Seveso, Italy.

In July, 1976 an explosion occurred at a chemical manufacturing site releasing a substantial amount of TCDD into the atmosphere. Contamination of plants, animals, and top soil occurred at this time. Evacuation of the area was not begun until 28 hours after the contamination occurred. It is estimated that 30,000 area residents were exposed. As also outlined in Appendix 3, initial skin effects included burns and contact dermatitis. A few weeks following the exposure 193 individuals, of whom 88% were children under 15 years of age, were diagnosed as having chloracne, a skin condition characterized by comedones, black heads, and epidermal cysts. Initial effects also included changes in liver function tests and elevated triglyceride levels (Pocchiari et al., 1979).

After ten years of follow-up (Assenato 1989), all but one of the 193 individuals had recovered from chloracne. Blood triglyceride and liver function tests were no different from the control population. These findings differ from previous studies in occupationally exposed groups where chloracne, abnormal liver function, and elevated triglyceride level have been noted many years after exposure (May 1982, Suskin 1984, Moses 1984). After 10 years of follow-up this study has not yet shown a detectable increase in birth defects or cancer.

Although reassuring, 10 years is a relatively short period of time to discover a long-term cancer hazard. Thus the final answer to whether this accidental exposure will

eventually lead to an increased cancer risk in the surrounding community remains unknown.

5.2.3 Polychlorinated biphenyls (PCB's): Controversies associated with PCB's have led to their almost daily appearance in the news media. Concern regarding the toxicity of PCB's surfaced following episodes of ingestion of PCB contaminated cooking oil in Japan (Kuratsune 1976) and Taiwan (Hsu et al., 1984). As also outlined in Appendix 3, at that time individuals were found to develop chloracne, liver and lipid disorders, peripheral neuropathy, and women pregnant at that time bore fetuses with birth defects. Health effects at that time were not found to be associated with blood PCB levels. The toxicity seen was later attributed not to the PCB's but to contaminating furans produced when the PCB oil was heated (Masuda and Yoshimua, 1984). Occupational groups exposed to PCB's have shown liver and lipid abnormalities but as yet no conclusive evidence that they are a cause of human cancers has been identified. One report combining all of the studies of PCB exposed workers suggests that there is an increased risk of liver, gallbladder and biliary tract cancer (IDSP 1987). PCB's are known to cause liver tumours in rodents (Ito et al., 1973; Kimborough et al., 1974; Kimborough et al., 1975). In addition, as they are almost indestructible, PCB's will persist in the environment for many years.

It is now well known that PCB's convert to much more dangerous compounds when burnt at relatively low temperatures. On February 5, 1981 an explosion occurred in the electrical panel in the basement of a Binghamton New York State office building, causing approximately 200 gallons of transformer fluid containing PCB's and chlorinated benzenes to be burned. When burnt these materials convert to dibenzofurans and dioxins (Schecter, 1985). One hundred and forty seven firemen and other persons who were in the building for 24 hours or more completed a medical surveillance program one year after the fire. Exposure to these contaminants were not found to cause substantial absorption of PCB's or any major short term health effects (Fitzgerald et al., 1986).

5.2.4 Pesticides: Pesticides is a generic term used to cover a variety of compounds which kill pests. It includes but is not limited to insecticides, herbicides, fungicides, and algacides. As such pesticides are made up of an extremely large number and variety of

chemical compounds. Depending on the class of compounds pesticides may either be broken down quickly or slowly in the general environment. Exposures to high doses of pesticides, either by inhalation, ingestion or skin contact may lead to neurologic, respiratory, or other health problems. As some pesticides are mutagens and carcinogens exposure to lower doses of these compounds may cause cancer or birth defects. The exact level of risk is dependent on the compound in question and the duration and extent of exposure. Recent evidence suggesting that phenoxy herbicides can cause cancer has emerged (Hardell, 1981; Hoar, 1986). The reader is referred to the numerous texts and reviews to explore the toxicity of specific pesticides. Appendix 3 and 4 should also be perused in this regard.

TABLE 5
RISKS OF DEATH FROM VOLUNTARY AND
INVOLUNTARY EXPOSRUES*

SOURCE OF EXPOSURE	LIFETIME AVERAGE RISK**	
Accidents		
All home accidents	7.7×10^{-3}	1 in 130
Lightning	3.5×10^{-5}	1 in 28571
Fire arms	7.0×10^{-4}	1 in 1429
Motor vehicles	1.7×10^{-2}	1 in 59
Electrocution	3.7×10^{-4}	1 in 2703
Cancers		
All cancers	1.9×10^{-1}	1 in 5
Buccal cavity, pharynx, and respiratory	5.0×10^{-2}	1 in 20
Digestive organs and peritoneum	5.3×10^{-2}	1 in 19
Bone, connective tissue, skin and breast	2.2×10^{-2}	1 in 45
Genital organs	2.2×10^{-2}	1 in 45
Urinary tract	8.4×10^{-3}	1 in 119
Leukemia and other blood and lymph	1.8×10^{-2}	1 in 56
Radiation		
Airline pilot (50 hr/mnth t 12 km altitute)	2.8×10^{-3}	1 in 357
One transcontinental round trip by air per year	7.0×10^{-5}	1 in 14286
Frequent airline passenger (4 h/week flying)	7.0×10^{-4}	1 in 1428
Average diagnostic medical x-rays	1.4×10^{-3}	1 in 714
Natural background radiation	1.4×10^{-3}	1 in 714
Living in masonry building rather than wood	3.5×10^{-4}	1 in 2857
Eating and Drinking		
One 12-1/2 ounce diet drink per day	7.0×10^{-4}	1 in 1429
Average saccharin consumption	1.4×10^{-4}	1 in 7143
Four tablespoons peanut butter per day	5.6×10^{-4}	1 in 1786
One pint milk per day	1.4×10^{-4}	1 in 7143
Miami or New Orleans drinking water	7.0×10^{-5}	1 in 14286
1/2lb charcoal broiled steak per week (cancer risk only)	2.1×10^{-5}	1 in 47619
Alcohol, one beer per day	1.4×10^{-3}	1 in 714
Tobacco		
Smoker, cancer only	8.4×10^{-2}	1 in 12
Smoker, all effects (including heardt disease)	2.1×10^{-1}	1 in 5
Person sharing room with smoker	7.0×10^{-3}	1 in 143
Air Pollution		
Polycyclic organics, all effects	1.1×10^{-3}	1 in 909

* From Travis and Cook (1989)
** All statistics based on U.S. data

CHAPTER 6

RIGHTS OF A COMMUNITY: TO KNOW, TO PARTICIPATE, TO BENEFIT DIRECTLY

As discussed by Tarr (1985), attitudes towards waste and methods of disposal are affected by a host of cultural, technical and economic factors. Normally wastes are rejected for health or asthetic reasons, but perspectives concerning what is or is not objectionable have shifted markedly over time. What is believed to be a health danger in one period of time or in one culture may be viewed differently in another time or by another culture; what one community sees as a nuisance to be eliminated, another community may fear, and yet another may perceive with indifference. The fundamental issue here is attitudes towards risk in regard to waste from either a health or socially related perspective.

Table 5 outlines risks associated with everyday life activities as calculated by Travis and Cook (1989) based on U.S data. These range from an average smoker's lifetime risk of death from smoking of 21% (or more than 1 in 5) to the average risk of death from lightning of .000035% (or about 1 in 30,000). However, in general, risks that are perceived as "voluntary" are accepted much more readily, and at a much higher level, than risks which are perceived as "involuntary". Within the involuntary category, risks that are perceived as "natural" are accepted more readily than risks which are perceived as "man-made". As such, the technical information regarding the actual level of risk is only one factor that must be considered in whether or not this risk is "acceptable".

Edelstein (1988) has provided an analysis of the psychological basis for the "not in my back yard" (NIMBY) syndrome. Over the past decade, there have been numerous examples of communities resisting the siting of "stigmatized" facilities, including landfills, hazardous waste disposal sites of all types, resource recovery plants, various sludge and septic facilities, nuclear waste disposal sites and nuclear power plants, potentially hazardous industries, microwave towers, and powerlines. In fact, similar resistance is also found regarding socially stigmatized facilities, including low income housing, trailer parks, and group homes for the mentally ill, aged, previously incarcerated, handicapped or even ill. Indeed one community in Winnipeg apparently rejected the establishment of a residence for students from the Professional Division of the Royal Winnipeg Ballet. While

it is inconceivable that the community feared that these dancers would be a health hazard in the community, the concerns that allowing such a group home would devalue their property was very real.

NIMBY, in the hazardous waste context, is a legacy of the publicity surrounding the Love Canal and other failures to apply safe disposal practices. NIMBY, unfortunately, is not a value-free term. As discussed by Edelstein (1988), while NIMBY may consist of conscious concern, narrow selfishness, or logical critique of an ill-conceived plan, government officials that use "NIMBY" tend to refer to this as an irrational rejection of sound technological progress. Because NIMBY implies that people are uninformed, the term "LULU" (Locally Undesirable Land Use) may be preferable. Others see NIMBY as a fight for democratic rights more properly termed "NIMBI" ("Now I Must Become Involved").

There is indeed a rational basis for opposing potential hazards. As Edelstein explains, three questions influence the assessment by concerned citizens of the legitimacy of a hazardous waste management proposal. The first of these questions is: "Are my concerns being heard and addressed?". It is contended that a major contribution to the anxiety of community members concerned about a proposed facility is the concern that they may not be able to participate directly in a decision that may greatly affect them. If the siting of a facility is to be accepted, it is important that the public is not disenfranchised in the process.

The second question is: "How will this project affect me?" While experts may be criticized that they employ the assumption that the facility designs will work as planned and therefore problems are unlikely to develop, citizens generally assume that if an impact is possible, it will occur. As such, this report and others, should be based on a worst-case analysis, as failure to do so would be seen by a community as failing to recognize that there are legitimate concerns.

The third question is: "Why me?". The fact that one community is asked to bear the risks for others without sharing the benefits provides a sufficient basis for perceptions of inequality. As the past has seen examples of undesirable projects situated in the poor and most disenfranchised segments of a community, it is understandable that

people may view the selection of their community as failing to acknowledge the worth of the community. In order to counter this potential feeling of inequality, it is necessary that the community perceive an actual benefit from the facility being established in their community. As such, it is not sufficient that a community be given access to all information, to fulfill the community's right to know; it is not sufficient that communities be allowed to participate in the decision making processes; the community must be actually empowered, and in a position to benefit, from the proposed landuse.

As concluded by Edelstein (1988) and others, opposition to the siting of a hazardous waste management facility may stem partly from concerns regarding health effects, however it also stems largely from the failure of regulators in the past to have taken seriously the possible psychosocial impacts of the choice of a community. Ironically, where the projects have developed quietly, with little public awareness and media involvement, there has been little psychosocial disruption in the community; it is only when a controversy develops, and then becomes highly publicized, that the community acquires the potential of becoming torn by different factions developing. Nonetheless the solution does not lie in further rationalizing the process, although firm scientific grounds for siting decisions are crucial. Instead, openness, communication, and most importantly, empowerment, ie. providing the community with real decision-making authority are the keys to minimize this disruption to a community's integrity. Indeed a community's self-image may be enhanced by the process and by the facility itself. It has been suggested (Edelstein, 1988), for example, that if the criteria that had been assumed to be met during siting are later found to have been violated, the community should be able to force the facility to close down as an automatic condition of its permit and acceptance by the community. Such guarantees, along with the opportunity for local citizens in communities to police the permit, including involvement of their own chosen experts, would help to give local residents a sense of control over the hazardous waste management facility.

As stated by Edelstein (1988), the goal is for vigilant citizenry, but not one that blindly opposes all projects dealing in risk. Thus, although this report had as its mandate to address the health aspects of the disposal of waste chemicals, the importance of the psychosocial process is such that it must be part-and-parcel of all interactions between a

community and the proponents of the hazardous waste management facility. The actions of the Manitoba HWMC to date indicate awareness of this reality.

CHAPTER 7

CONCLUSIONS AND RECOMMENDATIONS

Hazardous waste has, by definition, the potential to have negative effects on human health. However, in order to cause harm, exposure, in sufficient enough quantities, must occur. A great deal of information is available about adverse health effects possibly attributable to unmanaged or inadequately managed toxic waste disposal sites. Much of this information is equivocal and lacking in statistical significance, some is inferential, some indicates that no adverse effects are detectable. Little information is available about management sites where appropriate current technology is in use. To the extent that management of waste is successful in minimizing exposure potentials, little to no health effects should occur.

In a critical review Milham (1988) has commented that waste dumps should be cleaned up "because they stink and are eyesores,... rivers should be cleaned up because trout streams are nicer than open sewers." Few would argue with these sentiments and even in the absence of firm scientific conclusions it would be prudent to proceed on the basis of political common sense and to design hazardous waste management sites to the highest standards of current practice because unmanaged wastes are incomparably more unsightly, more undesirably and in all probability, more hazardous to the community than those which are appropriately managed.

Even with optimal use of waste reduction, reuse and recycling, the need for state-of-the-art hazardous waste management is uncontestable. While the health risk of a hazardous waste management facility is not zero, it is not thought to be high, in comparison with risks of everyday acceptable activities of life.

Recommendations:

Two themes characterize this report. First, is the theme of uncertainty. There is uncertainty related to health risk due to the past inadequacy of establishing exposure assessments and definition of exposed and unexposed populations, measurement of health outcomes, and determination of exposure-health outcome relationships. In order to minimize this uncertainty in the future, both for the community in question, as well as

for international knowledge on the question, the following suggestions could be considered by the community in question and the Hazardous Waste Management Corporation:

1) Computerized data banks should be established to detail the exact nature of waste materials entering this site. To the extent that this is feasible, these materials should be coded by actual chemical constituents so they could be linked to toxicological databases.

2) Environmental monitoring is an essential element of a control program at a hazardous waste management facility. The strategies for sampling of air, soil and water for select chemicals needs to be developed fully and communicated.

3) A comprehensive occupational health program for employees of the proposed facility is essential. Consideration could also be given to determining baseline exposure levels in other segments of the population with greatest potential for exposure to chemicals likely to be found at the management site. As biological monitoring is technically difficult and extremely expensive, a select list of priority chemicals would have to be established if such an activity is to be undertaken at all. Recognizing the limitations of such endeavors, an alternate possibility is banking specimens for possible future analyses if specific exposures ever become a focus of concern. Testing in future would undoubtedly be facilitated by the existence of baseline levels for select chemicals, however considerable further investigations would be required to assess the desirability, usefulness and feasibility of this endeavor.

The lack of factual information on health outcome or even hospital or physician visits, laboratory procedures, etc. have been a major hindrance to health risk assessments in the past. Manitoba is in a unique position to conduct health assessments utilizing the Manitoba Health Services Commission Database which records all lab procedures, as well as hospital and physician visits, by diagnosis. As such:

4) Consideration should be given to establishing a "health services utilization" profile for the community in question, to serve as a baseline upon which future analysis can be conducted.

As it is recognized that not all individuals attend physicians for non-specific symptoms or health concerns, questionnaires or door-to-door health assessment surveys are sometimes recommended. Although this may be considered by the community and the Hazardous Waste Management Corporation, this team of investigators is uncertain at the moment as to the need for this activity, given that it is a fairly expensive undertaking from which it is unlikely that useful information will be obtained.

The second major theme in the report is that of psychosocial impact of the facility on the dynamics of a community. The following is therefore recommended:

5) It is necessary that a community advisory group exist and remain active in the community in question; this group must be given whatever resources are necessary to conduct its own "policing" of the facility and its development.

6) All research materials (including all articles references in this report), all technical material, and any other requested by the community or any member of the community at any time in the future, must be made available at no expense to the community.

7) When a community decides to host the hazardous waste management facility, recognition of the social value of this facility to the province and region should be forthcoming and the community appropriately rewarded.

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GLOSSARY OF TERMS

Acute: (of exposure) usually short-term, sometimes resulting in illness; (of illness) immediately obvious, often severe (compare to chronic)

Ambient Standards: the officially accepted air levels in various substances in the workplace or in the environment.

Aquifer: an underground water-bearing geological formation

Aromatic Hydrocarbons: organic compounds containing primarily hydrogen and carbon, in which the molecules are arranged in closed rings typified by the benzene molecules (C_6H_6)

Bioconcentration: the increasingly greater concentration of a substance in an organism higher up in the food chain, i.e., in an animal that feeds on plants or other animals which have a lower concentration of that substance in their bodies. Also called bioaccumulation or biomagnification.

Carcinogen: a cancer-causing agent.

Chlorinated Solvents: organic solvents in which chlorine replaces one or more of the hydrogen atoms. Examples include dry-cleaning fluids such as perchlorethylene, and degreasers such as trichlorethane.

Chromosomal Aberrations: deviations from normal or typical development of chromosomes in the parts of cell nuclei responsible for determining and transmitting hereditary characteristics.

Chronic: (of exposure) long-term, often to small amounts of a substance; (of illness) of long duration, often not immediately apparent.

Comedo: a thickened secretion plugging a duct of the skin.

Cytogenetic: relating to the formation and development of cells.

Deposition: (of substances in the body) intake and accumulation.

Dioxins: a group of chlorinated aromatic hydrocarbons, some of which are highly toxic.

Energy Recovery Facilities: facilities where energy is recovered - i.e., heat generated by a treatment process is used to heat buildings. Many municipal incinerators burn garbage which is used to generate steam for heat or electrical generation. These are often referred to as energy recovery facilities.

Epidemiological: (of investigations) studies using statistical methods to collect and evaluate information about the distribution and determinants of diseases or public health related factors.

Hazardous Waste: waste which, if improperly managed, can create a risk to the safety or health of people or the environment; refers to products and substances outlined in The Dangerous Goods Handling and Transportation Act and its Regulations. Includes toxic waste.

Immunological: relating to the study of inherited, acquired or induced resistance to disease.

Incinerator: an apparatus that burns waste at high temperatures, under controlled conditions.

Ischemic: (of disease) lack of oxygen to body tissues produced by obstructions to blood flow.

Leach: to remove from a substance by the action of a percolating liquid; dissolve and drain away part of the contents.

Leachate: the liquid (usually rainwater) that, having percolated through a landfill, often contains materials dissolved from the landfilled waste.

Metabolites: products of the chemical and physical processes in a living organism.

Mortality: death rate; frequency of number of deaths in proportion to a population.

Occupational: of, relating to, or caused by an activity serving as one's employment.

Parity: a term used to designate the number of births (if any) that a woman has given.

Pesticides: chemicals used to kill pests, especially weeds (herbicides) or insects (insecticides).

Polychlorinated Biphenyls (PCBs): group of very stable chlorinated aromatic hydrocarbons, formerly used in a variety of applications, mostly in electrical equipment. Currently banned, mainly because of their persistence in the environment.

PPB: parts per billion; a unit of concentration

PPM: parts per million; a unit of concentration

Sister Chromatid Exchange: A process occurring during cell division; instrumental in the transfer of genetic information.

Solubility: the capability of being dissolved

Surface Impoundments: open pits, ponds and lagoons used for storing liquids

Toxic Substances: substances (chemicals) which are capable of producing injury, illness or damage to humans, domestic livestock, wildlife or other organisms through ingestion, inhalation, or absorption through any body surface.

Toxic Waste: poisonous waste substances; legally, products and substances referred to in Class 6 (Acute Toxic and Infectious) of the Dangerous Goods Handling and Transportation Act and its Regulations. All toxic waste is hazardous, but not all hazardous waste is toxic.

Trace metals: metals occurring in small amounts, often as impurities; sometimes toxic

ug/dl: microgram per 100 ml

Vapour Pressure: the pressure exerted by a vapour (i.e. the gaseous state of a substance) in equilibrium with its solid or liquid phase. Volatile compounds are characterized by higher vapour pressure

Volatility: the capability of a solid or liquid to quickly vaporize

Scope of Work

This Work will constitute the first Phase of the Corporation's proposed risk assessment program as outlined in Figure 1. It will provide the necessary background information to convey health/risk information to the public and assist in the future understanding of the overall risk assessment program. The proposed scope of work as outlined below, is intended to focus this investigation on public health issues surrounding hazardous waste management facilities. It should also be noted that Communities inviting us to pursue site selection work will have the opportunity to input into this scope of work and the overall risk assessment program as it is undertaken.

This investigation will be conducted as two Tasks. Task 1 will include a literature search and compilation of articles pertinent to this subject. Task 2 will include a critical review of this literature and the preparation of a summary report for public distribution.

The recommended scope of work for each Task is discussed briefly below:

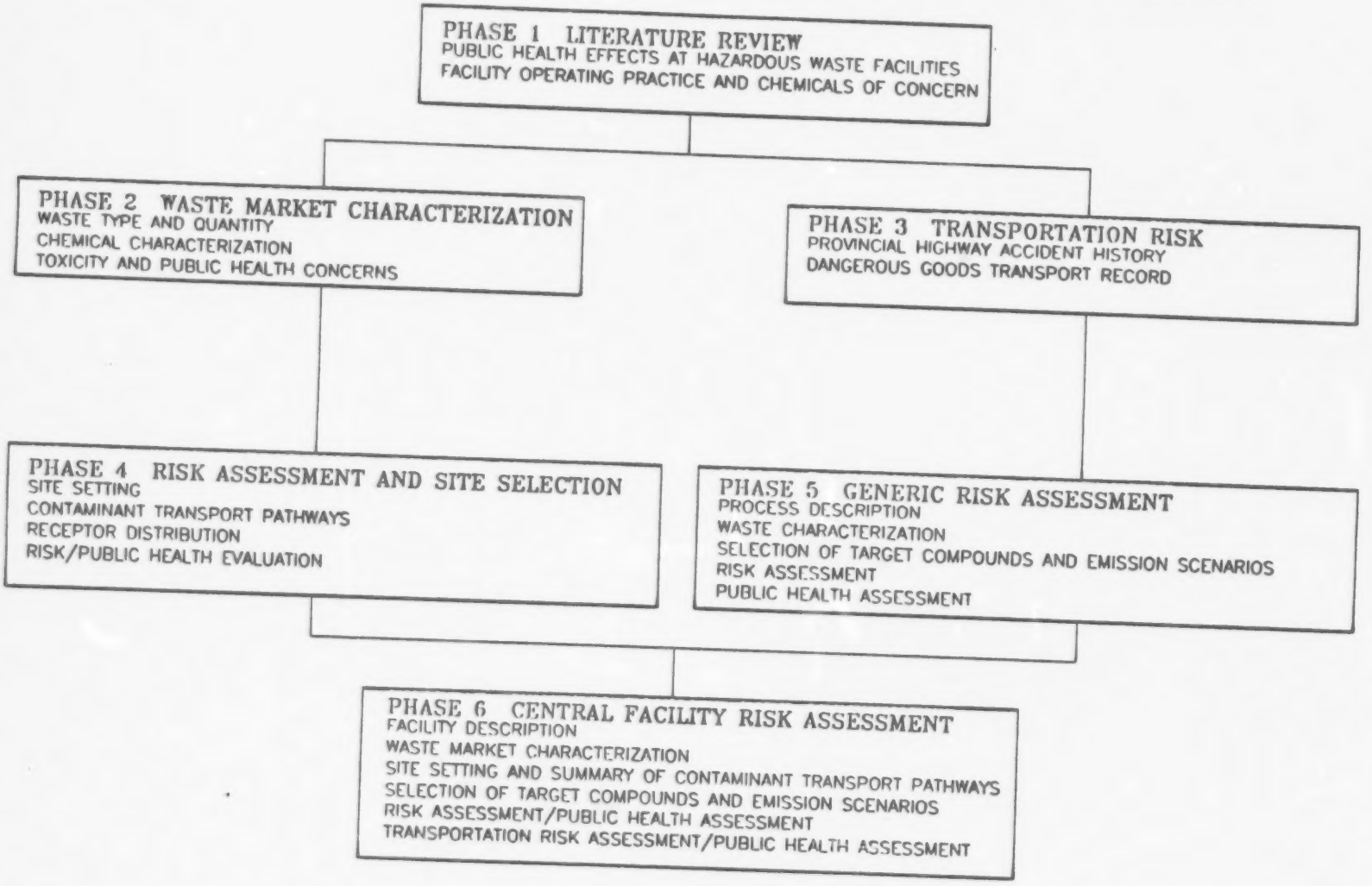
Task 1

The world literature pertaining to public health effects at existing hazardous waste treatment, storage and disposal facilities will be conducted. Based on recent comprehensive reviews of this subject (eg. Grisham, 1986, OWMC, 1986) this search will concentrate on literature published within the last five years. Although the literature review should concentrate on public health and exposure issues, the literature pertaining to occupational exposure could also be addressed. Copies of all pertinent research, resource and review articles will be obtained for critical review in Task 2.

Task 1 will also include a summary of potential public health exposure pathways associated with similar industrial facilities. This summary will help place exposure pathways into perspective and will assist in the definition of primary pathways to consider in the risk assessment.

All collected information will be reviewed for the purpose of defining the scope of work and cost estimate to complete Task 2.

FIGURE 1
RISK ASSESSMENT/PUBLIC HEALTH ASSESSMENT PROCESS


PHASE 1 LITERATURE REVIEW
PUBLIC HEALTH EFFECTS AT HAZARDOUS WASTE FACILITIES
FACILITY OPERATING PRACTICE AND CHEMICALS OF CONCERN
PHASE 2 WASTE MARKET CHARACTERIZATION
WASTE TYPE AND QUANTITY
CHEMICAL CHARACTERIZATION
TOXICITY AND PUBLIC HEALTH CONCERNS
PHASE 3 TRANSPORTATION RISK
PROVINCIAL HIGHWAY ACCIDENT HISTORY
DANGEROUS GOODS TRANSPORT RECORD
PHASE 4 RISK ASSESSMENT AND SITE SELECTION
SITE SETTING
CONTAMINANT TRANSPORT PATHWAYS
RECEPTOR DISTRIBUTION
RISK/PUBLIC HEALTH EVALUATION
PHASE 5 GENERIC RISK ASSESSMENT
PROCESS DESCRIPTION
WASTE CHARACTERIZATION
SELECTION OF TARGET COMPOUNDS AND EMISSION SCENARIOS
RISK ASSESSMENT
PUBLIC HEALTH ASSESSMENT
PHASE 6 CENTRAL FACILITY RISK ASSESSMENT
FACILITY DESCRIPTION
WASTE MARKET CHARACTERIZATION
SITE SETTING AND SUMMARY OF CONTAMINANT TRANSPORT PATHWAYS
SELECTION OF TARGET COMPOUNDS AND EMISSION SCENARIOS
RISK ASSESSMENT/PUBLIC HEALTH ASSESSMENT
TRANSPORTATION RISK ASSESSMENT/PUBLIC HEALTH ASSESSMENT

Task 2

Task 2 consists of the critical review of all materials collected as a result of the literature search and the preparation of a summary document addressing, at a minimum, the following:

- the types of facilities and/or hazardous waste management practices which have contributed to public health related concerns/impacts;
- the classes of hazardous wastes commonly associated with public health concerns/impacts;
- a critical review of world epidemiological and public health literature pertaining to public health effects at existing hazardous waste management facilities; and
- preparation of a public document addressing public health issues related to the development of a hazardous waste management facility in Manitoba.

A final document will be prepared which summarizes the results of the investigation. This report will be prepared for public distribution and the study team should be available to publicly address the results.

APPENDIX 2

SUMMARY OF METHODS AND FINDINGS OF SELECTED INVESTIGATIONS FOR HEALTH CONSEQUENCES RESULTING FROM CHEMICAL EXPOSURE OF POPULATIONS AT CHEMICAL DISPOSAL SITES*

TE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
Love Canal, NY	Odours and health complaints by residents	a)Vianna, 1980	82 Organic chemicals and metals; Inhalation; ingestion; contact in targeted exposure zones	Reproductive outcomes	Cohort study by questionnaire	460 live births	Excess of spontaneous abortions and low birth weight infants in wet areas, but no dose-response gradient.
		b)Picciano, 1980	as above, Residence near site	Chromosome breakage	Pilot study, no contemporary controls	36 resident volunteers, historic controls	No overall increase in aberrations; supernumery acentric fragments
		**c)Janerich et al. 1981	as above, Inhalation; ingestion	Cancer incidence	Retrospective cohort by census tract	225 households with 700 people in a census	Respiratory cancer rate higher than N.Y.State, but consistent with Niagara area, no excess liver cancer, lymphoma or leukemia.
		**d)Heath et al. 1984	as above, Detection of high airborne levels in basements	Chromosome abnormalities	Cohort study with matched controls	46 exposed (17 persons previously tested, 29 persons in targeted zones).	No increase in aberrations (including acentric fragments or sister chromatic exchange (SCE)
		**e)Bender and Preston, 1983	as above	Cytogenetic abnormalities; reproductive outcomes	Blinded cohort study with matched controls	46 exposed residents	No significant difference in chromosomal aberrations or SCE

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SITE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
		**f)Vianna and Polan, 1984	as above, contact in targeted exposure zones	Reproductive outcomes	Retrospective follow-up	1201 females, 617 births	Low birth weight observed in swale area residents during period of active dumping only.
		**g)Cuddy et al. 1984	As above, all subjects from historically wet areas	Theophylline clearance (liver function test)	Pilot study	11 resident volunteers, 25 controls	No significant differences.
		**h)Paigen et al. 1985	As above, exposure in utero	Low birth weight, prematurity and birth defects	Cohort study by questionnaire, stratified by homeowners vs renters	293 subjects, 707 controls	Low birth weight, increased in exposed homeowners but no renters; no increase in prematurity; birth deformations and malformations increased in exposed homeowners; malformations no increased in exposed renters.
		**i)Paigen et al. 1987	As above, children living in area at least 75% of lifetime	Height and weight	Cohort study	493 children, 428 controls	No weight difference, but significant shorter structure in subjects.
		**j)Polednak and Janerich, 1989	9 census tracts containing 12 disposal sites including Love Canal	Lung cancer	Pilot case-control study using mail questionnaires to assess exposure	339 cases, 676 controls; based on cancer registry	No increase in lung cancer

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SITE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
2. Velsicol Chemical Corp, Hardeman County, TN	Odours and health complaints by residents	**Clark et al. 1982; **Meyer, 1983	CC14, chlorobenzene, chloroform, hexachlorobi-cyloheptadiene, tetrachloroethylene; Ingestion of tainted well water; measurement of carbon tetrachloride in well water; indoor air sampling	Health questionnaire; clinical examination; laboratory tests	Case-comparison	82 exposed residents	Transitory liver injury was documented
3. Drake Chemical, PA	Worker complaints; community; concern for bladder cancer; EPA monitoring	**Budnick et al. 1984, **Grisham 1986	As above	Cancer mortality	County cancer mortality and reproductive	Based on census data and cancer registries;	Increase in bladder cancer but environmental (ie. beyond occupational) exposure to a bladder carcinogen
4. Woburn, MA	Leukemia cluster noted by local physician and state health agency resident complaints	a)Parker and Rosen, 1981	Pb, As, TCE Ingestion of tainted well water;	Childhood leukemia; renal, liver and bladder cancer	Case-comparison: 12 childhood leukemias; 20 kidney and 5 liver cancers	Census tracts, geographically targeted interviews	Cluster; excess of male kidney cancer and leukemia; no association to chemical exposure
	Leukemia cluster; study conducted to support law suit of families	**b)Byers et al. 1988	as above, members of 8 families with leukemic children		Lab and clinical studies of families with leukemic child; no controls	25 family members	Clinical and lab evidence of immunological damage, as well as neurological and cardiac damage

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SITE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
5. Lowell, Mass	Odours and health complaints by residents	**Ozonoff et al. 1987	Mixed chlorinated and aromatic hydrocarbons predominantly, proximity of residence	Symptom prevalence	Telephone survey of self-reported complaints	1049 interviews from 557 target households, 948 from 576 control households	Increased respiratory and general complaints
6. McColl, CA	Odours and health complaints by residents	**Lowrence 1981, Satin et al. 1983	Oil, sludge, sulfur compounds; targeted odour zones	General health; consultation of physicians and use of medical care facilities; cancer; reprodutive outcomes; pet health	Mailed questionnaires survey of 2 neighborhoods	1024 adults, 448 children 82% response rate from suspect neighbourhood compared to 69% response from compassion	No excess cancers, no impairement of intellectual skills in children, and no "serious medical problems"; eye and respiratory complaints, increased physician visits by children and excess menstrual complaints by women
7. Silresim, MA	Odours and health complaints by residents	Ozonoff et al. 1983	Oil, solvents, metals, PCB, pesticides; targeted airborne exposure zones	General health; consultation of physicians and use of medical care facilities; adults/children cancer incidence; mortality records; reproductive outcomes	Cross sectional survey: telephone and in-person interviews	1970 adults, 1100 children	No significant differences between exposed and control residents for cancer, reproduction, or mortality; greater frequency of respiratory symptoms, headache, fatigue, and heart problems

APPENDIX 2

SUMMARY OF METHODS AND FINDINGS OF SELECTED INVESTIGATIONS FOR HEALTH CONSEQUENCES RESULTING FROM CHEMICAL EXPOSURE OF POPULATIONS AT CHEMICAL DISPOSAL SITES*

SITE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
		**Lagakos et al., 1984	as above	Childhood leukemia reproductive outcomes; childhood disease	Blinded phone health survey questionnaire	3,257 homes	Exposure associated with childhood leukemia, perinatal mortality, birth defects, childhood disorders
	eport; well testing	**c.f.Grisham 1986	TCE; Ingestion of tainted well water	Bladder and liver disorder; skin rash; loss of equilibrium	Case incident report	750 families	One young girl with bladder and liver disorders
9. Montague, MI	Odours and health complaints by residents	**c.f. Grisham 1986	Chloroform, CCl4, TCE, Mirex, TCDD, As, hexachloro-cyclopentadiene; Ingestion of tainted well water and game fish	General health	Case incident report	12 homes	Residents report headache, nausea. No known latent effects.
10. Perham, MN	Hospital report of disease cluster	Crosslands, 1977; Feinglass, 1983	Arsenic trioxide; Ingestion of tainted well water; As levels in hair; estimation of water consumption	Neurologic and GI tract function	Case-comparison	13 homes	Gastrointestinal distress and neuropathy were consistent with exposure to arsenic.

APPENDIX 2

SUMMARY OF METHODS AND FINDINGS OF SELECTED INVESTIGATIONS FOR HEALTH CONSEQUENCES RESULTING FROM CHEMICAL EXPOSURE OF POPULATIONS AT CHEMICAL DISPOSAL SITES*

SITE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
11. "GEMS" landfill, Gloucester Environmental Management Services, NJ	Odours and health complaints by residents	**c.f.Grisham, 1986	Toluene, organic solvents; targeted airbone exposure	General health; consultation of physicians and use of medical care facilities; respiratory function; reproductive outcomes	4 Cross-sectional cohort surveys; medical questionnaire	566 exposed residents; 164 controls	No excess medical or reprodutive problems; excess respiratory symptoms, eye irritation, and odour complaints among the exposed group; no significant differences in pulmonary function
12. Jackson Township Landfill, NJ	Health complaints by residents	New Jersey Health Dept	Benzene, chloroform TCE, acetone, methylene chloride Ingestion of tainted well water	General health; cancer; skin; reproductive outcomes; nervous system; blood; respiratory; GI tract.	Case incident report	96 residents	Skin ailments; complaints of kidney ailments, cancer and mis-carriages.
			as above; Ingestion of tainted well water; measurements of volatile organics in water and indoor air	Skin; reproductive outcomes	Cross-sectional; health census	560 exposed residents	Increased incidence of skin complaints
13. Krysowaty Farm, NJ	Resident complaints of health problems; EPA monitoring	N.J. Dept. of Health	Benzidine, benzene hexachloro-benzene, heavy metals; Ingestion of tainted well water; duration of use of well water	12 Subjective symptoms; pregnancy; consultation of physicians and use of medical care facilities	Cross-sectional survey	12 exposed households; 30 control households	Increased tiredness for females; questionable increase for miscarriages; no significant differences between exposed and unexposed for a variety of subjective complaints

APPENDIX 2

SUMMARY OF METHODS AND FINDINGS OF SELECTED INVESTIGATIONS FOR HEALTH CONSEQUENCES RESULTING FROM CHEMICAL EXPOSURE OF POPULATIONS AT CHEMICAL DISPOSAL SITES*

SITE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
14. Pomona Oaks, NJ	Resident complaints of foul well water	N.J. Dept. of Health	Benzene, volatile organics; indoor air measurements; urinary phenol measurements	Potential exposure to a known carcinogen; GLOBAL 82 risk assessment model**	Prospective cross-sectional survey	153 residents	GLOBAL 82 model estimated an increased cancer risk from inhalation of benzene at levels in excess of acceptable.
15. Price Landfill, NJ	Contaminated groundwater	N.J. Dept. of Health	Benzene, chloroform TCE, vinyl chloride toluene, Pb, Zn, Cd; Ingestion of tainted well water from proven plume of contaminated aquifer	General health; consultation of physicians and use of medical care facilities; reproductive outcomes	Cross-sectional cohort	121 exposed residents; 123 controls	Excess muscle pain, rash and skin irritation, joint pain, pregnancy problems in exposed group; no long-term effects; (symptoms deemed possibly stress-related)
16. Reich's Farm, Dover Township NJ	Resident complaints of foul well water	Ghassemi, 1976 **Grisham, 1986	Acetone, toluene, styrene, CC14 methyl ethyl ketone; Ingestion of tainted well water	Medical complaints; illness	Illness survey	23 families (48 persons)	No correlation between use of contaminated well water and reported illness
17. "Rutherford," Universal Oil Products and Chemical Processing Inc., NJ	Resident concerns for cluster of leukemia and Hodgkin's disease	Burke et al. 1988 Halperin et al. 1988	Benzene, TCE; Inhalation; residents living within the school district	Incidence of leukemia and Hodgkin's disease	Case-comparison interview questionnaire	13 cases leukemia; 9 cases Hodgkin's disease	Increased incidence of leukemia in age group 5-19 and Hodgkin's disease in age group 20-39; no significant exposure source consistently associated with all cases

APPENDIX 2

SUMMARY OF METHODS AND FINDINGS OF SELECTED INVESTIGATIONS FOR HEALTH CONSEQUENCES RESULTING FROM CHEMICAL EXPOSURE OF POPULATIONS AT CHEMICAL DISPOSAL SITES*

SITE	REASON FOR STUDY	REFERENCES	PREDOMINANT CHEMICALS AND EXPOSURE CRITERIA	MAJOR HEALTH ENDPOINTS	STUDY DESIGN	SAMPLE SIZE	FINDINGS REPORTED
18. Brookfield Landfill, NY	Odour complaints by residents	Schultz, 1983	Non-specific; targeted airborne exposure zones	Subjective symptoms; utilization of medical care facilities	Cross-sectional telephone and interview survey	555 households (454 exposed, 101 control)	Statistically significant increase in resident complaints and physician visits, especially for children
19. "Hooker Hyde Park," NY	Citizen complaints of odours and skin irritation	**Rothenberg 1981, Singal et al. 1982	TCDD, many chlorinated hydrocarbons; Residence or employment near landfill; pesticide blood levels	Physical examination of skin, mucous membranes, and thyroid; blood pressure; laboratory tests of urine and blood	Cross-sectional community study, interview questionnaire, and NHANES data comparisons	428 persons (residents and workers near landfill)	No indicators of ill health were attributable to the waste site
20. ARM Wade, PA	Complaints of acute symptoms following fire at site	Falk et al. 1980	Benzene and naphthalene deriatives, Cr, Pb, Ni, Zn, Cu Inhalation; direct contact; targeted exposure zones	General health; consultation of physicians and use of medical care facilities; respiratory symptoms; skin rashes; child health profile with clinical laboratory tests	a)Non-random door-to-door survey; b)local physician interviews c)firemen questionnaire	86 adults; 28 children (14 children with medical examination); 35 firemen	No history of adverse health effects on residents or from physical examination of 14 children; firemens' complaints of headache, cough and skin rashes could not be conclusively related to this fire

NOTE: * This table is largely adapted from Grisham, 1986. See Grisham for refrences.
** These references are also available from the authors at the Occupational and Environmental Health Program, University of Manitoba.

APPENDIX 3

INCIDENTS OF ACCIDENTAL ENVIRONMENTAL OR INDUSTRIAL EFFLUENT CHEMICAL EXPOSURE*

Chemical and Circumstance of Exposure	Reference	Study Design	Measure of Exposure	Study Findings
A) ORGANIC CHEMICALS				
1) Phenol: Wisconsin, 1974: Railroad accident led to spill of phenol. Contaminated well water consumed by residents.	**a)Baker et al., 1978	Case-comparison Exposed: N=39 Comparisons: N=119 Blinded study; clinical exam and lab tests performed without knowledge of exposure status.	Indirect: Concentration of phenol in well water and proximity of residence	Acute symptoms of diarrhea, sores and dark urine, burning of the mouth occurred only in exposed. Clinical findings 6 months after the incident revealed significant differences between exposed and comparisons.
2) 4,4'-Diaminodiphenylmethane (4,4'-DAPM): U.K., 1965: Flour was contaminated by a spilled canister of 4,4'-DAPM during transport by truck.	**a)Kopelman et al., 1966	Case series: N=84	Indirect: All patients had eaten bread from the the bakery to which the contaminated flour had been delivered.	Abdominal pain, fever and generalized aches, enlarged liver, and jaundice with increased bilirubin. Symptoms ranged from mild to severe.
3) Methyl isocyanate (MIC): Bhopal, India, 1984: MIC, used as intermediate in pesticide production, accidently released from industrial source into air in highly populated area.	a) Anonymous, 1984	News reports. No epidemiological studies yet conducted due to recency of the incident.	Direct: ? Indirect: proximity to cloud of MIC gas released.	More than 2,000 deaths due to acute pulmonary edema. An estimated 20,000 to 100,000 people sustained injuries involving lung damage, skin burns, and eye ulcerations. Incident too recent for possible chronic effects to be observed.
4) Ortho-cresyl phosphate (OCP): Bombay, 1960: Contaminated mustard oil used for cooking. Means of contamination was not established	**a) Vora et al., 1962	Case series: N=58 (32 examined in detail)	Indirect: Patients had purchased oil from several shops from which OCP-contaminated oil samples were later collected.	Acute peripheral polyneuritis, primarily affecting distal segments of limbs. Pain and paralysis, followed by "glove and stocking" sensory impairment. Characteristic high-stepping, lurching gait. Occasional bradycardia, fever and diarrhea.
Morocco, 1959: OCP-contaminated olive oil consumed by residents was supplied by a single wholesaler to shops in all affected areas.	**a) Smith and Spaulding, 1959	Case series: N>2000	Indirect: Self-reported history of consumption of dark oil purchased from shops supplied by wholesaler who distributed contaminated oil	Acute peripheral polyneuritis affecting distal segments of limbs. Pain and parasthesia followed by "glove and stocking" sensory impairment. Characteristics high-stepping, lurching gait. Occasional bradycardia,

Chemical, Route of Exposure, Circumstances	Reference	Study Design	Measure of Exposure	Study Findings
*Chemical species unspecified, however, effects noted in study findings are consistent with effects known to be produced by Tri-ortho-cresyl phosphate (TOCP).				fever, and diarrhea.
5) Acrylamide: Japan 1974: Acrylamide used in construction of a sewage system near patients' residence. Well water used for drinking and bathing was contaminated.	**a) Igisu et al., 1975	Case series: N=5	Indirect: High concentrations of acrylamide found in well water.	CNS disturbances--subacute mental confusion and truncal ataxia, followed by polyneuropathy. More severe in 3 adults than in 2 children. Liver enzyme levels and various blood blood tests were normal
6) 2,3,7,8-Tetrachlorodi-benzo-p-dioxin (TCDD): Missouri, 1971: TCDD was a contaminant in waste oils sprayed on residential, recreational, and work areas for dust control.	a) Stehr et al., 1984	Case-comparison Exposed: N=68 Comparison: N=36 Blinded study; clinical exam and lab tests performed without knowledge of exposure status	Indirect: Residence or employment in exposed areas, or high rate of contact with contaminated soil.	No significant differences between exposed and comparisons in hepatic, dermatologic, immunologic, and neurologic status. Authors noted that this should be considered a pilot study.
Italy, 1976: An industrial explosion dispersed TCDD over a large area.	a) Cardanoni and Fara, 1982	Survey: Residents in the vicinity of the plant	Indirect: Proximity of residence to the plant	Higher frequency of dermatologic problems in residents nearest the plant, but no gradient beyond areas of closest proximity.
		Case-comparison: Cases: N=163 Comparisons: N=51 Study attempted to correlate the presence or absence of chloracne with exposure.	Indirect: Residence in contaminated area during and after the explosion, animal deaths, eating own or neighbors' garden products, playing or working outdoors.	No differences between cases and comparisons in exposure history.
	b) Sirchia et al., 1982	Cohort: Exposed children: N=48 Unexposed comparisons: different subjects at each follow up screening. Tests of immunologic function were performed blind (without knowledge of exposure status)	Indirect: Proximity of residence to plant.	Several test results (e.g. total serum complement hemolytic activity, and lymphocyte response to mitogens) showed higher values for exposed than for unexposed children. Results were not anticipated since TCDD experiments in animals suggest an immunosuppresant effect. Suggests that perhaps the effects noted are due to immunostimulation, or that misclassification of exposure status occurred.
	c) De Carli et al., 1982	Cohort: Examined women who had induced abortions. Exposed: N=13 Unexposed comparisons: N=12	Indirect: Proximity of residence to plant	Frequency of chromosomal aberrations was significantly higher in fetal tissue from exposed than from unexposed. No difference observed for the other tissues.

Chemical, Route of Exposure, Circumstances	Reference	Study Design	Measure of Exposure	Study Findings
		Examined maternal blood, placenta, umbilical cord and fetal tissue.		
		Cohort: Acute exposure: N=145 Chronic exposure: N=69 Unexposed comparisons: N=87	Indirect: Proximity of residence and duration of time spent in plant.	No statistically significant differences in frequency of chromosomal aberrations.
7) Polychlorinated biphenyls (PCB) Japan 1968: Rice oil used for cooking was contaminated by PCB during manufacture.	**a) Kuratsune et al., 1972	Case series: N=189 adults	Indirect: Self-reported use of rice oil, nearly all of which was produced or shipped by one company during a specific period of time	Disease called "Yusho." Brown pigmentation of nails and skin, acneform skin eruptions. Increased eye discharge, swelling of upper eyelids. Feeling of fatigue and weakness. Reproductive--13 births to exposed mothers. 2/13 were stillborn; 10/13 showed unusually stained skin pigmentation.
	**b) Kuratsune et al.,1976			More recent studies suggest formation of polychlorinated dibenzofurans (PCDF) during heating of contaminated oil may have had a role in causation of Yusho.
Taiwan, 1979-1980: Rice oil contaminated by PCB.	a) Chen et al., 1980	Case studies N>1900	Direct: Serum PCB levels	Epidemic of an undescribed "peculiar skin disease". Reported to be similar to 1968 Japanese "Yusho" outbreak as described in #9 above.
Alabama, 1979: PCB found in environmental samples (sediments, water, fish) downstream from a chemical plant.	a) Kreiss et al., 1980	Survey: A majority of residents in the community were examined. N=458	Direct: Serum PCB levels	Serum PCB levels were positively correlated with serum cholesterol, gamma GT, and borderline and definite hypertension. No correlation with SGPT and total bilirubin. No adverse reproductive events were associated with serum PCB levels.
Indiana, 1960's-70's: PCB-contaminated sewage sludge used for fertilizer.	**a) Baker et al., 1980	Survey: Four groups with varying exposures were evaluated. Exposed: N=126 Comparisons: N=22	Direct: Serum PCB levels.	No chloracne or symptoms of toxicity. No correlations between serum PCB levels and tests of hematologic, hepat or renal function. Serum PCB correlated a positively with plasm triglycerides suggesting that PCB may alter lipid metabolism.

Chemical, Route of Exposure, Circumstances	Reference	Study Design	Measure of Exposure	Study Findings
8) PBB: Michigan, 1973: Packaging mix-up resulted in PBB being mixed with cattle feed. Contamination of dairy animals resulted in widespread human exposure.	a) Landrigan et al., 1979	Cohort: Six cohorts with varying exposures identified: 3 groups at risk for high exposure: N=3820 3 groups with low level exposure: N=725.	Direct: Serum PBB levels	No association found between any symptom prevalence rates and serum PBB levels. No statistically significant differences in lymphocryte number or function. The cohort identified is to be followed prospectively for chronic disease.
	**b) Bekesi et al., 1978	Case-comparison: exposure: N=45	Direct: Serum PBB levels	Those with serum PBB levels had increased numbers of "null" cells (lymphocytes with no detectable surface markers), decreases in lymphocytes that form rosettes with sheep erythrocytes, and altered lymphocyte function. No consistent correlation between serum PBB concentration and altere lymphocytes, however, no unexposed subject showed such abnormalities.
B) ORGANIC PESTICIDES				
1) DDT: Alabama, 1978-1979: Downstream from a pesticide plant, high DDT metabolite residues detected in fish consumed by community residents.	**a) Kreiss et al., 1981b	Survey: N=499	Direct: Serum DDT levels.	No acute health effects were demonstrated. Serum DDT levels positively associated with serum cholesterol, triglycerides, and -GT. No correlation with SGPT and total bilirubin levels. Study did not address possible long-term effects.
2) Endrin: Egypt, 1966: Bread was made from treated seed intended exclusively for planting. Bread made from flour contaminated with endrin was eaten. Contamination assumed to occur during transport or storage.	**a) Coble et al., 1967	Case series: N=4	Direct: Serum or urine endrin levels in 3 or 4 cases. Indirect: History of contaminated bread consumption in 4 of 4 cases.	3 of 4 persons who ate contaminated bread were affected. CNS--headache, dizziness, confusion, convulsions. Nausea and vomiting. Recovery was rapid and complete.
U.K., 1956: Endrin had leaked onto the floor of a wagon and was absorbed by sacks of flour being transported at a later date Flour was subsequently baked into bread.	a) Davies and Lewis, 1956	Case series: N=59	Indirect: All cases purchased bread from the one bakery which had received contaminated flour.	In mild cases, dizziness, disorientation, weakness in legs, accompanied by nausea. In severe cases (30 subjects), convulsions of sudden onset. Severity of effects related directly to amount of contaminated bread consumed. Recovery was fairly rapid and apparently complete.

Chemical, Route of Exposure, Circumstances	Reference	Study Design	Measure of Exposure	Study Findings
3) Hexachlorobenzene (perchlorobenzene): Turkey, 1955-1959: Wheat seed treated with the fungicide hexachlorobenzene was used for consumption rather than for planting, its intended purpose.	**a) Cam and Nigogosyan, 1963	Case series: N=348 Cases in a region of Turkey were examined in the hospital. Total number of people affected has been estimated at N>3000.	Indirect: History of consumption of treated seed intended for planting, or residence in area where treated seed was distributed.	Porphyria cutanea tarda due to interference with porphyrin metabolism in the liver. Symptoms manifested most severely in child primarily in males, and were characterized by: -Blistering and ulceration of skin -Hypertrichosis and hyperpigmentation with depigmented scars. -Corneal opacities -Atrophy of hands and muscles -Red urine with porphyrinuria -Weight loss and anorexia, but no abnominal pain. No neurological or mental disturbances observed. Symptoms exacerbated by exposure to sunlight. Acute skin manifestations disappear after discontinuation of intake, but relapses may occur during summer months.
4) Parathion: Singapore, 1959: Barley became contaminated with parathion at some unestablished point during shipping from Europe.	**a) Kanagaratnam et al., 1960	Case series: N=53 Persons reported to local hospitals for medical care.	Indirect: Cases purchased and consumed barley from several shops, all of whose barley came from one consignment from importer.	38 of 53 exposed were affected. 9 children died. In severe cases, collapse and coma, accompanied by sweating, muscle fasiculation, excessive salivation, and dyspnea due to increased lung secretions Constricted pupils. In mild cases, vomiting and diarrhea headache and malaise. All who survived recovered completely.
Jamaica, 1976: Flour contaminated by parathion at unestablished point during transport from Europe.	**a) Diggory et al., 1977	Case series: N=79 Persons reported to local hospitals.	Direct: Paranitrophenol found in urine of 9 of 15 cases. Indirect: History of ingestion of baked goods from contaminated flour.	Acute organophosphate poisoning in which 17 of 79 cases died. In mild cases, nausea, vomiting and pinpoint pupils Severe cases resulted in convulsions stupor or coma, accompanied by excessive salivation, diplopia, dyspnea muscle fasciculations, and bradycardia. Depressed plasma or RBC cholinesterase levels observed.

Chemical, Route of Exposure, Circumstances	Reference	Study Design	Measure of Exposure	Study Findings
C) METALS				
1) Arsenic: Oregon, 1958-71: Naturally occurring high levels of arsenic in well water.	**a) Morton et al., 1976	Cross-sectional correlation: Census tract rates of skin cancer	Indirect: Residence in tracts with high arsenic levels in drinking water.	No relationship between rates of skin cancer (basal cell and squamous cell carcinomas) and arsenic levels in drinking water, although arsenic levels were lower than in other studies where positive correlations have been found Mean arsenic level in rural areas was 16.5 ppb (range 0-2150 ppb), and in urban areas was 4.8 ppb (range 0-860 ppb).
Taiwan, 1965: Naturally occurring high levels of arsenic in water from artesian wells, in use since 1900.	a) Tseng, 1977	Survey: N=40,421 Inhabitants of the area of endemic high arsenic levels	Indirect: Arsenic concentrations in the well water of study subjects.	Manifestations of chronic arsenicism were skin cancer, hyperpigmentation, and keratosis. Also, blackfoot disease, a local term for a peripheral vascular disorder resulting in gangrene of the extremities. A clear dose-response gradient was observed between consumption of arsenical water and development of each of the above disorders. Arsenic levels in well water ranged from 10 to 1820 ppb.
Chile, 1968-71: Naturally occurring levels of arsenic in city water supply. Reduced by filtration plant in 1970.	a) Zaldivar, 1974	Case series: N=457 Cases presented to the regional hospital for evaluation.	Direct: Arsenic content of hair. Indirect: Residence in area served by water supply with high level of water	Manifestations of chronic arsenic poisoning were: cutaneous lesions (including hyperkeratosis and squamous cell carcinoma), Raynaud's bronchitis, weight loss and general debility. Thrombosis and myocardial infarctions were prominent events in children. Mean arsenic level in drinking water was 600 ppb (range 50-960 ppb).
2) Cadmium: Japan, 1950's-60's: Cadmium discharged into a river by a chemical plant. Downstream water used for drinking and irrigation of rice fields.	a) Friberg et al 1974.	Surveys and case-control studies.	Direct: Blood and urine levels of calcium and phosphate. Indirect: Residence in endemic area; estimated cadmium intake.	Major symptoms of "Itai-itai (ouch-ouch) disease: ·Skeletal deformity, leg and lumbar back pain, multiple fractures. ·Proteinuria, glucosuria, renal tubular dysfunction. ·Osteomalacia with increased serum alkaline phosphate and decreased serum inorganic phosphate. Deficient nutritional status in conjunction with cadmium poisoning may be a contributing etiological factor.

APPENDIX 4
ADVERSE HEALTH EFFECTS OF CHEMICALS BY
HUMAN TARGET ORGAN*

TABLE A: EFFECTS ON THE SKIN

A) SKIN PIGMENT

EFFECT	CHEMICAL
Hyperpigmentation (darkened colour)	Arsenic Bismuth Mercury Silver
Hypopigmentation (loss of skin pigment)	Dihydroxybenzenes Catechol, p-tert-butyl Catechol, p-isopropyl- Catechol, p-methyl Hydroquinone Hydroquinone, monobenzyl ether Hydroquinone, monoethyl ether Phenol, amyl- Phenol, p-tert-butyl- Phenyl, nonyl- Phenol, octyl- Physostigmine (eserine) N,N',N"-triethylenethio-phosphoramide (Thio-Tepa)
Pigment discoloration	Organic chemicals Acid, picric Acid, oxalic Dinobuton (2-1-methyl-2-propyl)-5,6-dinitrophenyl isopropylcarbonate 4,4'-Methylenedi-aniline p-Phenylenediamine Inorganic chemicals Nitric acid Sodium nitrate Permanganate Metals Arsenic salts Bismuth salts Copper salts Lead salts Mercury salts Silver nitrate

* Adapted from Buffler et al, 1985
References are cited in full in Buffler et al, 1985.

APPENDIX 4
ADVERSE HEALTH EFFECTS OF CHEMICALS BY
HUMAN TARGET ORGAN

TABLE A: EFFECTS ON THE SKIN (cont'd)

B) CONTACT DERMATITIS

CHEMICAL	IRRITANT	SENSITIZER (ALLERGEN)
Organic chemicals		
Acetone	X	
Acid, acetic	X	
Acid, benzoic	X	X
Acid, butyric	X	
Acid, cinnamic	X	
Acid, cresylic	X	
Acid, formic	X	
Acid, lactic	X	
Acid, oxalic	X	
Acid, picric	X	
Acrylic monomer	X	
Alcohol, amyl		X
Alcohol, benzyl		X
Alcohol, butyl	X	X
Alcohol, ethyl	X	X
Alcohol, propyl		X
Adehyde, cinnamic	X	
Amide, oleyl		X
Amine, monoamyl		X
Aminophenazone		X
Aminothiazole		X
Aniline	X	X
Benzene	X	
Benzene, dichloro-	X	X
Benzene, dinitrochloro-	X	X
Benzoate, sodium	X	
Benzophenone	X	X
Carbon disulfide	X	X
Diamine, phenylene	X	X
Diamine, toluene	X	X
Formaldehyde	X	X
Lindane		X
Nitro and nitroso compounds		X

APPENDIX 4
ADVERSE HEALTH EFFECTS OF CHEMICALS BY
HUMAN TARGET ORGAN

TABLE A: EFFECTS ON THE SKIN (cont'd)

CHEMICAL	IRRITANT	SENSITIZER (ALLERGEN)
Phenazone, amino	X	X
Phenol	X	
Phenols, chlorinated	X	
Phenylhydrazine	X	X
Polyamides, aliphatic		X
Polyethylene glycol		X
Polysorbate		X
Propionate, phenylmercuric		
Thiazole, amino		X
Toluamide, diethyl		X
Toluene, butylhydroxyl		X
Trichloroethylene	X	X
Trinitrotoluene		X
Inorganic chemicals		
Acid, chromic	X	X
Acid, hydrofluoric	X	
Acid, nitric	X	
Acid, sulfuric	X	
Ammonium compounds	X	
Bromides	X	
Calcium oxide	X	
Calcium carbonate	X	
Calcium hypochlorite	X	
Cyanide, sodium or potassium	X	
Silicate, sodium	X	
Metals and their salts		
Antimony salts	X	
Arsenic salts	X	X
Arsenic insecticides	X	
Cobalt chloride	X	
Mercury salts	X	X
Nickel oxide	X	X
Nickel salts	X	X
Platinum salts	X	X

APPENDIX 4
ADVERSE HEALTH EFFECTS OF CHEMICALS BY
HUMAN TARGET ORGAN

TABLE A: EFFECTS ON THE SKIN (cont'd)

C) FOLLICULITIS AND ACNEFORM

EFFECT	CHEMICAL
Folliculitis and acneform dermatosis (e.g. chloracne)	Polyhalogenated naphthalenes Polychloronaphthalenes Polybromonaphthalenes Polyhalogenated biphenyls Polychlorinated biphenyls (PCBs) Polybrominated biphenyls (PBBs) Polyhalogenated dibenzofurans Polychlorodibenzofurans, especially tri-, tetra-, penta-, and hexachloro-dibenzofurans; Polybronodibenzofurans, especially tetrabromodibenzofuran Contaminants of polychlorophenol compounds especially herbicides (2,4,5T and penta-chlorophenol) and herbicide intermediates (2,4,5T-Trichlorophenol) 2,3,7,8-Tetrachlorodibenzo-p-dioxin Hexachlorodibenzo-p-dioxin Tetrachlorodibenzofuran Contaminants of 3,4-dichloroaniline and related herbicies (Propanil, Methazole) 3,4,3',4'-Tetrachoroazoxybenzene 3,4,3',4'-Tetrachloroazobenzene 2,6-Dichlorobenzonitrile
Porphyria cutanea tarda	Chlorinated hydrocarbon intermediates
Aopecia (hair loss)	Chloroprene
Neoplasms	Chloroprene Soots, tars, mineral oils Arsenic

APPENDIX 4
TABLE B
ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS REPORTED TO BE DUE TO CHEMICALS: NERVOUS SYSTEM EFFECTS

EFFECTS	CHEMICAL
Behavioral changes	Organic chemicals
Neurasthenia, irritability, impaired psychomotor function, and memory impairment	Organophosphate insecticides Perchloroethylene Solvents, especially Acrylamide Styrene Toluene Xylene
	Inorganic chemicals
Acute psychosis, emotional instability, memory impairment	Carbon disulfide
	Metals
Neurasthenia, irritability	Arsenic
Neurasthenia, irritability, emotional instability, memory impairment	Lead
Neurasthenia, irritability, acute psychosis, emotional instability, memory impairment	Manganese
Neurasthenia, irritability impaired psychomotor function	Mercury
Central nervous depression	Organic chemicals
	Ethyl alcohol
	Aliphatic alcohols, ketones, aldehydes
	Alkenes, including butadiene
	Alkanes (C_1-C_6)
	Aromatic hydrocarbons (benzene toluene, xylene)
	Methylene chloride
	Trichloroethane
	Inorganic chemicals
	Carbon disulfide
Peripheral neuropathies	Organic chemicals
	Acrylamide
	DDT
	Hexane
	Methyl n-butyl ketone
	Methyl chloride
	Solvents
	Trichloroethylene
	Inorganic chemicals
	Carbon disulfide
	Metals
	Arsenic
	Lead
	Manganese
	Mercury
Ataxic gait	Organic chemicals
Myoclonus	Acrylamide
Ataxic gait	Benzene hexachloride
Ataxic gait, opsoclonus, tremors	Chlordane
Ataxic gait, tremors	Chlordecone (Kepone)
Bladder neuropathy	Chlorophenothane (DDT)
Ataxic gait, impaired visual acuity	Dimethylaminopropionitrile
Impaired visual acuity	Hexane
Ataxic gait	Methanol
Seizures	Methyl n-butyl ketone
Palsy, Guillain-Barre syndrome	Organochlorine insecticides
Atax gait	Organophosphate insecticides
Cranial neuropathy	Toluene
	Trichloroethylene
	Inorganic chemicals
Acute psychosis, cranial neuropathy, headache	Carbon disulfide
	Metals
Seizures Parkinsonism	Lead
Tremors, headache	Manganese
Ataxic gait, myoclonus, tremors, impaired visual acuity, inttracranial pressure	Mercury
Neoplasm	Vinyl chloride
	Chlordane, heptachlor

APPENDIX 4

TABLE C

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS REPORTED TO BE DUE TO CHEMICALS: LIVER EFFECTS

EFFECTS	CHEMICAL
Acute hepatocellular injury (hepatitis)	Organic chemicals
	Chlorinated benzenes
	Biphenyls, polychlorinated (PCB)
	Carbon tetrachloride
	Chlordecone (Kepone)
	Chloroform
	Dioxin
	Ethylene dibromide
	Ethylene dichloride
	Naphthalenes, chlorinated
	Nitrosamine, dimethyl
	Inorganic
	Phosphorus compounds
	Metals
	Arsenic
	Copper
Subacute necrosis and cirrhosis	Organic
	Biphenyls, polychlorinated (PCB)
	DDT
	Tetrachloroethane
	(perchlorethylene)
	Toluene
	Trinitrotoluene
	Vinyl chloride
	Metals
	Arsenic
Cirrhosis	Organic
	Biphenyls, polychlorinated (PCB)
	Carbon tetrachloride
	Hexachlorobenzene
	Naphthalene, chlorinated
	Metals
	Beryllium
Neoplasms	
	Carbon tetrachloride
	2,3,7,8-Tetrachlorodibenzo-p-dioxin
	Vinyl chloride
	Arsenic

APPENDIX 4

TABLE D

ADVERSE HEALTH EFFECTS REPORTED TO BE DUE TO CHEMICALS

REPRODUCTIVE SYSTEM AND FETUS

EFFECTS	CHEMICAL
Germ cell mutation	Vinyl chloride (suspected)
Infertility, male (includes decreased spermatogenesis)	Organic chemicals Carbaryl (suspected) Carbon disulfide (suspected) DDT (cyclodiene group of chlorinated insecticides) Dibromochloropropane Epichlorohydrin (suspected) Kepone (suspected) Polychlorinated biphenyls (PCB) Chloroprene Metals Cadmium (suspected) Lead Manganese (suspected)
Infertility, female	Organic chemicals Benzene (suspected) Carbon disulfide (suspected) Chloroprene (2-chlorobutadiene)(syspected) Dibromochloropropane (suspected) Ethylene dibromide (suspected) Kepone (suspected) PCB (suspected) Metals Cadmium Lead
Embryotoxicity	Organic chemicals Benzene (suspected) PCB Metals Lead Arsenic (suspected) Mercury (suspected)

APPENDIX 4

TABLE D

ADVERSE HEALTH EFFECTS REPORTED TO BE DUE TO CHEMICALS

REPRODUCTIVE SYSTEM AND FETUS

EFFECTS	CHEMICAL
Teratogenesis (congenital anomalies/ malformations)	Organic chemicals
	Aldrin (suspected)
	Benzene (suspected)
	Captan (suspected)
	Carbaryl (suspected)
	Dimethyl sulfoxide (suspected)
	Dioxin (suspected)
	Ethylene dibromide (suspected)
	Ethylene thiourea (suspected)
	Hexachlorophene (suspected)
	Malathion (suspected)
	PCB
	Perchloroethylene (suspected)
	Propylene glycol (suspected)
	2,4,5-Trichlorophenoxyl acetic acid (suspected)
	Vinyl chloride (suspected)
	Metals
	Arsenic (suspected)
	Cadmium (suspected)
	Lead (suspected)
	Mercury
Spontaneous abortion/fetal death	Organic chemicals
	Chloroprene (suspected)
	Dibromochloropropane
	2,4,5-Trichlorophenoxyacetic acid (suspected)
	Dioxin (suspected)
	Ethylene dibromide (suspected)
	PCB
	Vinyl chloride (suspected)
	Inorganic chemicals
	Carbon disulfide (suspected)
	Metals
	Arsenic (suspected)
	Cadmium (suspected)
	Lead
	Mercury (suspected)
Neonatal death	Organic chemicals
	PCB (suspected)
	Metals
	Cadmium (suspected)

APPENDIX 4

TABLE D

ADVERSE HEALTH EFFECTS REPORTED TO BE DUE TO CHEMICALS

REPRODUCTIVE SYSTEM AND FETUS

EFFECTS	CHEMICAL
	Lead
	Mercury (suspected)
Low birth weight	Organic chemicals
	Chloroprene (suspected)
	Perchloroethylene (suspected)
	PCB
	Metals
	Cadmium (suspected)
	Lead
	Mercury (suspected)
Developmental disabilities	Organic chemicals
	PCB (suspected)
	Metals
	Arsenic (suspected)
	Cadmium (suspected)
	Lead
	Mercury
Childhood cancer (cancer in offspring)	Organic chemicals
	Hydrocarbons (suspected)
	Metals
	Lead

APPENDIX 4

TABLE E

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS REPORTED TO BE DUE TO CHEMICALS:

BLOOD-FORMING AND LYMPHATIC SYSTEM

EFFECTS	CHEMICAL
Anemia	Organic chemicals
	Nitrobenzene
	Naphthalene
	Naphthol
	Phenol derivatives
	Phenylhydrazine
	Quinones
	Metals
	Arsenic trioxide
	Arsine
	Copper sulfate
	Lead
Marrow depression	Benzene
	Lead
Immune suppression	Polychlorinated biphenyls (suspected)
Aplastic anemia	Benzene
	Benzene hexachloride (Lindane)
	Trinitrotoluene
Leukemia and lymphoma	Benzene
	Chlordane and heptachlor (uncertain)
	Chloroprene (uncertain)
	Dichlorobenzene (uncertain)
	Epichlorohydrin (uncertain)
	Ethylene oxide (uncertain)
	Hexachlorcyclohexane
	Trichloroethylene (uncertain)
	Vinyl chloride (uncertain)

APPENDIX 4

TABLE F

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS
REPORTED TO BE DUE TO CHEMICALS:
GENITOURINARY SYSTEM

EFFECTS	CHEMICAL
Acute renal failure	Organic chemicals
Prerenal ischemia	Aniline
Secondary effects of hemolytic agents	Carbon tetrachloride
Direct tissue injury	Chlordane
	Ethylene glycol
	Parathion
	Paraquat
	Toluene
	Trichloroethylene
	Metals
	Mercury
Chronic renal disease	Carbon disulfide
Tubal or interstitial disease	Cadmium
	Lead
Glomerulonephritis	Solvents
Neoplasia kidney	Cadmium (uncertain)
	Lead (uncertain)
Neoplasia, bladder	4-Aminobiphenyl
	Benzidine
	2-Naphthylamine
	N-Phenyl-2-naphthylamine (uncertain)
	Soots, tars, oils
	o-p-Toluidine
	Lead (uncertain)
Neoplasia, prostate	Cadmium (uncertain)

APPENDIX 4

TABLE G

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS REPORTED TO BE DUE TO CHEMICALS INCLUDING METALS:

LUNGS AND RESPIRATORY TRACT

EFFECTS	CHEMICAL
Asphyxiation	Organic chemicals
	Ethane (simple)
	Methane (simple)
	Inorganic
	Carbon dioxide (simple)
	Carbon monoxide (chemical)
	Hydrogen cyanide (chemical)
	Hydrogen sulfide (chemicals)
	Nitrogen (simple)
Irritation	Inorganic chemicals
	Ammonia
	Halogen gases
	H-X(any concentrated acid mist where X is a halogen)
	Nitrogen oxides
	Ozone
	Phosgene
	Sulfur oxides
Sensitization, asthma	Organic chemicals
	Anhydride, phthalic
	Anhydride, trimetallic
	Anhydride, tetrachlorophthalic
	Amine, phenylene-
	Chloramine
	Formaldehyde
	Isocyanates
	Phenol
	Piperazine formaldehyde
	Pyrethrins
	Sulfonechloramine
	Tannic acid
	Inorganic chemicals
	Isocyanates, inorganic
	Metals
	Chromium
	Cobalt
	Nickel sulfate
	Platinum salts (complexed)
	Vanadium pentoxide, trioxide
Hypersensitivity pneumonitis	Phthalic anhydride
	Trimetallic anhydride
	Toluene diisocynate
	Organic dusts

APPENDIX 4

TABLE G

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS REPORTED TO BE DUE TO CHEMICALS INCLUDING METALS:

LUNGS AND RESPIRATORY TRACT

EFFECTS	CHEMICAL
Pneumoconioses	Oil shale
	Minerals
	Asbestos
	Coal
	Silica
	Talc
	Metals
	Antimony
	Cerium oxide
	Tungsten carbide
Fibrosis	Asbestos
	Silica
Edema	Smoke (products of pyrolysis)
	Inorganic chemicals
	Ammonia
	Chlorine gas
	Nitrogen oxides
	Phosgene
	Metals
	Cadmium
Granulomatous disease	Inorganic chemicals
	Talc
	Metals
	Aluminum
	Beryllium
	Zirconium
Mesothelioma	Asbestos
Cancer	Organic chemicals
	Acrylonitrile (uncertain)
	Amitrole (uncertain)
	Bischloromethyl ether
	Chloroprene (uncertain)
	Epichlorohydrin (uncertain)
	Hexachlorocyclohexane (uncertain)
	Mustard gas
	Soots, tars, mineral oils
	TCDD
	Vinyl chloride
	Vinylidene chloride (some forms)

APPENDIX 4

TABLE G

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS REPORTED TO BE DUE TO CHEMICALS INCLUDING METALS:

LUNGS AND RESPIRATORY TRACT

EFFECTS	CHEMICAL
	Minerals
	Asbestos
	Hematite (uncertain)
	Metals
	Arsenic
	Beryllium (uncertain)
	Cadmium (uncertain)
	Chromium
	Nickel (some forms)

APPENDIX 4

TABLE H

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS:

GASTROINTESTINAL TRACT

EFFECTS	CHEMICAL
Esophagitis	Acids, mineral Alkalis
Gastritis, acute or chronic	Ethyl alcohol
Neoplasia, pharyngeal-esophegeal-laryngeal	Organic chemicals Formaldehyde Isopropyl oils Mustard gas Soots, oils, tars Metals, minerals Chromium (uncertain) Nickel (uncertain) Asbestos (uncertain)
Neoplasia, stomach	Organic chemicals Chloroprene (uncertain) Soots, tars, and mineral oils Vinyl chloride (uncertain) Minerals, metals Asbestos (uncertain)
Neoplasia, colon	Organic chemicals Acrylonitrile (uncertain) Chloroprene (uncertain) Ethylene oxide (uncertain) Soots, tars, and mineral oils Vinyl chloride (uncertain) Minerals, metals Asbestos (uncertain)

APPENDIX 4

TABLE I

ADVERSE HEALTH EFFECTS IN HUMAN TARGET ORGANS:

CARDIOVASCULAR SYSTEM

EFFECTS	CHEMICAL
Hypertension	Cadmium Lead (chronic)
Heart disease, indirect effect	Dinitro benzene Methylene chloride Trinitro toluene (TNT)
Heart disease, direct effect	Organic chemicals Fluorocarbons Ethylene glycol chlorinated hydrocarbons (some) Inorganic chemicals Carbon disulfide Metals Arsenic Cobalt

* Appendix 4 is adapted from tables in Buffler et al. (1985)

APPENDIX 5

CHEMICALS FOUND AT CHEMICAL DISPOSAL SITES THAT MAY EFFECT THE IMMUNE SYSTEM*

CHEMICAL (with code)	IMMUNE DISFUNCTION	CHEMICAL	SPECIES	SOURCE**
Aromatic Compounds	Decreased mitrogen transformation	(a)	mouse	Wierda et al., 1981
(a) Benzene	Decreased antibody production	(a)	mouse	Wierda et al., 1981
(b) Toluene	Decreased MM[1]	(d)	mouse	La Via and La Via, 1979
(c) Xylene	Impaired host resistance	(d)	rat	Exon and Koller, 1984
(d) Phenol	Increased susceptibility to tuberculosis	(a)	rabbit	WHO/IARC, 1982b
	Decreased IgG[2], IgA[3] serum levels	(a)(b)(c)	human	Lange et al., 1973
	Decreased complement serum levels	(a)(b)(c)	human	Smolik et al., 1973
	Lymphocytopenia	(a)	human	Aksoy, 1981
Halogenated Aliphatic Compounds	Increased mitogen transformation	(g)	mouse	Sharma and Gehring, 1979
(e) Chloroform (trichloromethane)	Decreased MMI	(f)(h)(i)	mouse	Munson et al., 1982
(f) 1,2-Dichloroethane (ethylene dichloride)	Decreased CMI[4]	(i)	mouse	Sanders et al., 1982
(g) Vinyl chloride (monochloroethylene)	Leukocytopenia	(f)	mouse	Munson et al., 1982
(h) Vinylidene chloride (1,1-dichloroethylene)	Impaired host resistance	(e)	mouse	Capel et al., 1979
(i) 1,1,2-Trichloroethylene	Increased embryonic antigen	(g)	human	Page et al., 1976
Halogenated Aromatic Compounds	Impaired host resistance	(j)(n)	mouse	Loose et al., 1978
(j) Hexachlorobenzene (HCB)		(l)	mouse	Thigpen et al., 1975
(k) Pentachlorophenol (PCP)		(l)	mouse	Luster et al., 1980
(l) 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)		(o)	mouse	Neilson et al., 1973
(m) 2,3,7,8-Tetrachlorodibenzofuran (TCDF)	Increased GVH[5] reaction	(n)	mouse	Carter and Clanoy, 1980
(n) Polychlorinated biphenyls (PCBs)	Thymus atrophy	(l)(n)	mouse	Vos et al., 1980
(o) 2,2-bis(p-Chlorophenyl)-1,1,1-trichloroethane (DDT)		(o)	mouse	Street and Sharma, 1974
	Decreased mitogen transformation	(l)	mouse	Luster et al., 1980
	Increased mitogen transformation	(n)	mouse	Silkworth and Loose, 1979
	Decreased MMI	(j)(n)	mouse	Loose et al., 1978
		(l)	mouse	Thomas and Hinsdill, 1979
		(n)	mouse	Thomas and Hinsdill, 1978
	Decreased CMI	(l)	mouse	Luster et al., 1980

APPENDIX 5

CHEMICALS FOUND AT CHEMICAL DISPOSAL SITES THAT MAY EFFECT THE IMMUNE SYSTEM*

CHEMICAL (with code)	IMMUNE DISFUNCTION	CHEMICAL	SPECIES	SOURCE**
		(l)	mouse	Clark et al., 1983
	Decreased complement activity	(o)	mouse	Street and Sharma, 1975
	Increased HMI	(k)	mouse	Anderson and White, 1984
	Decreased HMI	(j)	rat	Vos et al., 1979
	Increased antitumor activity	(o)	rat	Wasserman et al., 1969
	Impaired host resistance	(n)	rat	Kerkvliet and Kimeldorf, 1977
	Decreased antibody production	(k)	rat	Exon and Koller, 1984
	Impaired host resistance	(o)	chicken	Rao and Glick, 1977
	Thymus atrophy	(o)	chicken	Radhakrishnan et al., 1972
	Decreased CMI	(m)	guinea pig	Luster et al., 1979
	Decreased mitogen transformation	(m)	guinea pig	Luster et al., 1979
	Normal Ig complement levels	(m)	guinea pig	Luster et al., 1979
	Increased respiratory infection	(l)	human	Reggiani, 1980
	Decreased CMI	(n)	human	Shigematsu et al., 1978
		(n)	human	Chang et a., 1980
Trace Metals	Altered host resistance	(p)	mouse	Gainer and Pry, 1972
(p) Arsenic		(r)	mouse	Koller, 1980
(q) Asbestos		(t)	mouse	Cook et al., 1975
(r) Cadmium		(t)(u)	mouse	Gainer, 1977
(s) Chromium		(v)	mouse	Sobocinski et al., 1977
(t) Lead	Accelerated thymus atrophy	(u)	mouse	Hirokawa and Hayashi, 1980
(u) Mercury	Decreased HMI	(r)(t)	mouse	Koller et al., 1976
(v) Zinc		(u)	mouse	Koller et al., 1977
		(p)(t)(u)	mouse	Blakley et al., 1980
	Impaired macrophage function	(q)	mouse	White, 1981
		(t)	mouse	Blakley and Archer, 1981
		(v)	mouse	Karl et al., 1973
	Lymphocyte-macrophage binding	(q)	rat	Miller et al., 1979
	Increased GVH reaction	(u)	rat	Pelletier et al., 1983
	Decreased virus Ab[6] titer	(s)	rat	Figoni and Treagan, 1975
	Decreased ADCC[7]	(q)	human	Kagamimori et al., 1980
	Increased Ig serum levels	(q)	human	Kagan et al., 1977

APPENDIX 5

CHEMICALS FOUND AT CHEMICAL DISPOSAL SITES THAT MAY EFFECT THE IMMUNE SYSTEM*

CHEMICAL (with code)	IMMUNE DISFUNCTION	CHEMICAL	SPECIES	SOURCE**
	Decreased circulating T-cells[8]	(q)	human	Kagan et al., 1977
	Allergic contact dermatitis	(s)	human	Peltonen and Fraki, 1980
		(u)	human	Baer et al., 1973
	Impaired host resistance	(t)	human	Sachs, 1978
		(t)	human	Ewers et al., 1982
	Decreased HMI	(t)	human	Reigart and Graber, 1976
	Glomerulonephritis	(u)	human	Druet et al., 1982
Miscellaneous Organic Compounds	Decreased NK[9] cell activity	(x)	mouse	Luster et al., 1982
(w) Formaldehyde	Allergic contact dermatitis	(y)	guinea pig	Karol et al., 1980
(x) Ethyl carbamate (urethane)	Respiratory tract hypersensitivity	(y)	guinea pig	Karol et al., 1980
(y) Toluene diisocyanate (TDI)	Respiratory tract hypersensitivity	(y)	human	O'Brien et al., 1979
	Allergic contact dermatitis	(w)	human	Nardana, 1980

* From Grisham, 1986

** References cited in full in Grisham, 1986

THE TYPES OF IMMUNOLOGICAL ABNORMALITY ABBREVIATED ABOVE

1 Humoral mediated immunity
2 Immunoglobulin G
3 Immunoglobulin A
4 Cell mediated immunity
6 Antibody
7 Antibody-dependent cell-mediated cytotoxicity
8 Thymus derived cells
9 Natural killer cells
10 Graft versus host

APPENDIX 6
TABLE A
CARCINOGEN POTENCY VALUES
(INHALATION) FOR HAZARDOUS WASTE
CONSTITUENTS*

Carcinogenic constituent	Cancer Risk factor $(mg/kg/day)^{-1}$
Dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin	425,000
Bis(chloromethyl)ether	9,300
Aflatoxins	2,930
1,2-Dimethylhydrazine	803
Dibenzo(a,i)pyrene	476
Benzidine	234
Diethylstilbestrol	107
N-Nitrosodiethylamine	43
4-Nitroquinoline-1-oxide	40
Coal tars	31
Dieldrin	30
N-Nitrosodimethylamine	25
Arsenic and compounds	14
Benzo-a-pyrene	11.5
Aldrin	11.4
Hexachlorocyclohexane, Alpha isomer	11.1
Reserpine	10.4
Kepone	8.97
DDT	8.42
N-Nitrosodi-n-butylamine	5.43
Beryllium and compounds	4.86
Polychlorinated biphenyls	4.34
2,4-Diaminotoluene	4.03
Hydrazine	3.91
Heptachlor	3.76
Heptachlor epoxide (Alpha, Beta, Gamma)	3.37
N-Nitrosophyrrolidine	2.13
Hexachlorocyclohexane, Beta	1.84
3,3-Dichlorobenzene	1.69
Hexachlorobenzene	1.68
Chlordane	1.61
Lindane	1.33
Bis(2-chloroethyl)ether	1.14
Toxaphene	1.13
1,1-Dichloroethene	1.04
1,2-Diphenylhydrazine	0.77
Amitrole	0.74
Acrylonitrile	0.55
Methyl hydrazine	0.36
2,4-Dinitrotoluene	0.31
Pentachloronitrobenzene	0.26
1,1,2,2-Tetrachloroethane	0.20
Chloroform	0.18
Urethane-ethyl carbamate	0.15
Thiourea	0.14

APPENDIX 6
TABLE A (con't)
CARCINOGEN POTENCY VALUES
(INHALATION) FOR HAZARDOUS WASTE
CONSTITUENTS*

Carcinogenic constituent	Cancer Risk factor $(mg/kg/day)^{-1}$
Formaldehyde	0.083
Carbon tetrachloride	0.083
Hexachlorobutadiene	0.078
1,1,2-Trichloroethane	0.057
Benzene	0.052
1,1,2,2-Tetrachloroethene	0.040
1,2-Dichloroethane	0.037
Aniline	0.030
Safrole	0.025
2,4,6-Trichlorophenol	0.019
Vinyl chloride	0.017
Hexachloroethane	0.014
Ethylphthalyl ethylglycolate	0.014
Bis(2-ethylhexyl)phthalate	0.014
Trichloroethylene	0.013
1,4-Dioxane	0.010
N-Nitrosodiphenylamine	0.005

A cancer potency factor of 1.0mg/kg/day, for example, indicates that theoretically an individual exposed to 1 mg/kg for 70 years would get cancer (for the average 70 kg person, this would mean a daily exposure to 70 mg for seventy years). A potency of .001 indicates 1 case per 1000 individuals to 1 mg/kg for 70 years; a cancer potency value of 100 suggests that there would be 1 excess cancer for every person exposed to .01 mg/kg/day for 70 years. "Excess" means that the risk is in addition to the risk of cancer presented by other sources. The table illustrates that the cancer potency estimates cover a broad range of excess risk, seven orders of magnitude.

APPENDIX 6

TABLE B: ACCEPTABLE DAILY INTAKES (INHALATION) FOR HAZARDOUS WASTE CONTITUENTS, SORTED BY ASCENDING ADI

Noncarcinogenic constituent	ADI (MG/DAY)
Phorate	0.0024
Lead (Pb)	0.0045
4-Chlorophenol	0.0050
Acrolein	0.0090
Nitroglycerine	0.0095
Toluene diisocyanate	0.013
Sodium azide or smite	0.014
Silver and compounds	0.016
Phosgene	0.019
Methyl parathion	0.020
Mercury	0.020
Cyanogen chloride	0.029
Hexachlorocyclopentadiene	0.036
Thallium and compounds	0.037
Bromodichloromethane	0.039
Parathion	0.040
1,1-Dimethylhydrazine	0.048
Maleic anhydride	0.048
Endrin	0.070
Bis(2-chloroisopropyl)ethyl	0.070
4,6-Dinitro-o-cresol and salts	0.071
2,6-Dinitrotoluene	0.071
Hydrofluoric acid	0.071
Methyl ethyl ketone peroxide	0.071
Oxirane	0.095
Acrylamide	0.11
Methomyl	0.12
Chloroacetaldehyde	0.14
1,3-Dichloropropene	0.18
Pentachlorophenol	0.18
Benzyl chloride	0.24
Dinitrobenzene	0.28
1,3-Dinitrobenzene	0.28
1,2-Dinitrobenzene	0.28
1,4-Dinitrobenzene	0.28
Endosulfan	0.28
Crotonaldehyde	0.29
Phthalic anhydride	0.29
Antimony and compounds	0.29
Cyanides (soluble salts)	0.33
1,2,4,5-Tetrachlorobenzene	0.35
Diphenylamine	0.36
Tetranitromethane	0.38
Furfural	0.38
Tribomomethane	0.39

APPENDIX 6

TABLE B: ACCEPTABLE DAILY INTAKES (INHALATION)
FOR HAZARDOUS WASTE CONTITUENTS,
SORTED BY ASCENDING ADI

Noncarcinogenic constituent	ADI (MG/DAY)
Benzo(j,k)fluorene	0.42
Formic acid	0.43
1,2,4-Trichlorobenzene	0.46
2-(Chloromethyl)oxirane	0.48
Hydrocyanic acid	0.52
Selenium and compounds	0.70
Pyridine	0.71
2,4-Dimethylphenol	0.80
DDE	0.84
Dimethylamine	0.86
1,2-Dichloropropane	0.98
Chlorobenzene	1.01
Methoxychlor	1.40
Carbon disulfide	1.43
Acrylic acid	1.43
Bromomethane	1.45
Nickel and compounds	1.46
Ethylbenzene	1.60
Methyl ethyl ketone	1.72
Trichloronaphthalene	1.80
Di-N-butyl-phthalate	1.80
Dimethyl phthalate	1.80
Resorcinol	2.14
DDD	3.01
Nitrobenzene	4.00
Cyclohexanone	4.76
Hydrogen sulfide	5.00
Methyl methacrylate	5.23
2,6-Diaminotoluene	5.70
Phenol	6.80
2,4-Dichlorophenol	7.00
2,4,5-Trichlorophenol	7.00
1-Butanol	7.14
Isobutyl alcohol	7.14
Cresols	7.90
Acetaldehyde	8.57
2,4-Dinitrophenol	9.45
Methyl isobutyl ketone	9.76
Isophorone	10.50
Methanol	12.38
Dichloromethane	12.90
N-Phenylthiourea	13.70
Pentachlorobenzene	14.00
Zinc and compounds	15.00
Dichloropropane	16.66

APPENDIX 6

TABLE B: ACCEPTABLE DAILY INTAKES (INHALATION) FOR HAZARDOUS WASTE CONTITUENTS, SORTED BY ASCENDING ADI

Noncarcinogenic constituent	ADI (MG/DAY)
Naphthalene	18.00
Xylene	20.71
Acetonitrile	25.00
Tetrahydrofuran	28.09
Acetophenone	29.60
2,5-Diaminotoluene	30.30
Chloromethane	37.50
1,1,1-Trichloroethane	37.50
Cyclohexane	50.00
Ethyl ether	57.14
Ethyl acetate	66.66
Acetone	84.76
1,2-dichlorobenzene	107.00
Tetrafluoromethane	129.00

* Adopted from Travis and Cook (1989)